

SECTION OF HUMAN SKIN

(1) Epidermis (2) true skin (3) subcutaneous tissue (4) horny layer; (5) clear layer (6) granular layer (7) permatinative layer (8) capillary network, (9) artery (10) vein (11) lobules of fat (12) nerve (13) corpuscle of Vater (14) sweat gland (15) duct of sweat gland (16) pore of sweat gland (17) hair follicle (18) hair shaft (19) bulb of a hair (20) arrector pili (21) sebaceous gland (22) duct of sebaceous gland (White Laboratories, Inc.)

ESSENTIALS OF DERMATOLOGY

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FIFTH EDITION

211 Figures
11 Subjects in Color



Philadelphia

Montreal

J B LIPPINCOTT COMPANY

Fifth Edition

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Library of Congress
Catalog Card Number
56-6409

PRINTED IN THE UNITED STATES OF AMERICA

AFFECTIONATELY DEDICATED TO
AGNES B TOBIAS
AND MY DAUGHTERS
SALLY AND SUSAN

Preface to the Fifth Edition

In the interval since the previous edition the author has had the opportunity of evaluating the newer drugs and technics used in dermatology. In line with suggestions made by readers, the rarer dermatoses have been deleted, and more space has been given to the more common dermatoses. Their comments are greatly appreciated. Although syphilis is no longer an important part of dermatologic practice, the author does not think it wise to omit at this time the chapter dealing with the disease. While it is true that a lack of cases exists for teaching purposes, we do not know what the future has in store.

The number of photographs has been increased from 186 to 211. Some are from my own collection. Others have been generously contributed by the following: Drs. R. L. Howard, John C. Slaughter Jr., R. O. Noofin, Paul Mapother, H. R. Cogburn, V. Pardo-Castello, Beatrice H. Kuhn, E. A. Edwards, A. B. Loveman, M. T. Fliegelman, L. J. A. Lowenthal, Kathleen Riley, A. Marin, B. Usher, C. C. Barret, and Roy L. Kile.

Additional colored photographs have been supplied by the White Laboratories and the Abbott Laboratories, for which I am deeply grateful. Also in this edition will be found lists of instructions for patients with acne rosacea, pruritus and infantile and atopic eczema, and there are pertinent references which are intended to be helpful though not comprehensive.

By keeping the book concise and yet as comprehensive as possible, I hope physicians will continue to find it useful and convenient.

My deepest thanks are due Mr. Brooks Stewart and Mr. Stanley A. Gillet and the staff of the publishers for their many suggestions.

NORMAN TOBIAS

St. Louis, Missouri

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Basic Survey

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PREDISPOSING CAUSES	MICROSCOPIC EXAMINATIONS
EXCITING CAUSES	BLOOD SEROLOGIC TESTS FOR SYPHILIS
CUTANEOUS DIAGNOSIS	BIOPSY
INSPECTION	CUTANEOUS MANIFESTATIONS OF SYSTEMIC DISEASES
REGIONAL LOCALIZATION	FUNCTIONS OF THE SKIN
DETERMINATION OF THE PRIMARY LESION	ANATOMY AND PATHOLOGY OF THE SKIN
DETERMINATION OF SECONDARY LESIONS	EPIDERMIS
GROSS PATHOLOGIC CHANGES	CUTIS
PALPATION	SUBCUTANEOUS TISSUE
SUBJECTIVE SYMPTOMS	CUTANEOUS APPENDAGES
DIAGNOSTIC METHODS	EMBRYOLOGY OF THE SKIN
WOOD'S LIGHT	NURSING ASPECTS
SEROLOGIC TESTS	
INTRADERMAL TESTS	

DERMATOLOGY is that branch of internal medicine which consists of the diagnosis, the interpretation and the treatment of diseases of the skin. Investigation of the skin should not be limited to that organ rather the entire patient should be considered with special reference to the integument and its disorders.

A knowledge of the morphology and the characteristic localization of the various types of skin disorders, an adequate history and a proper routine examination are fundamentals in dermatologic diagnosis. Proficiency can be gained only by experience.

The skin has been aptly called the mirror of the body reflecting the imprint of disordered metabolism or physiologic balance. These disturbances may be brought about by numerous noxious influences acting from without or from within. As a result of these alterations in this complex cutaneous structure over 300

patterns of disease may be produced. To separate and classify the various cutaneous disorders, we make use of clinical knowledge, laboratory tests, serologic and immunity reactions, animal inoculations, bacteriologic and cultural investigation and tissue studies.

The history the etiologic factors the methods of cutaneous diagnosis and the functions and the anatomy of the skin will be discussed in this chapter

HISTORY TAKING

Beginners usually question the patient before looking. The experienced dermatologist looks first and then asks specific questions to confirm his diagnosis, to obtain facts suggestive of a possible cause for the lesion or the eruption, to discover the details of previous therapy etc. and to learn the patient's reaction to his disease. A routine history may point to the necessity of a more definite and detailed line of questioning. Inexpert questioning may bias the examiner's opinion or lead him astray. In any case the physician should be sympathetic with the patient's explanation of the cause of his dermatosis, be it diet, "acid blood poor circulation nervousness, a "bad liver" or weed poisoning. A regular order in recording the history and the results of the dermatologic and general physical examination is important from the standpoint of completeness and future reference.

The routine history for skin eruptions should cover the following points:

Name	Address	Age
Occupation	S M. D. W.	Race
Chief complaint	Duration	

Where did your eruption first appear?

What did it look like in the beginning? Has it changed its appearance?

Were you ill before the rash appeared?

State the order in which the various parts of the body were affected.

Is the rash spreading stationary or decreasing?

List in order the different applications and internal remedies you have used. Which ones were helpful useless or harmful?

Have you had a similar rash before? When? What helped it?

Has anyone in your family a similar condition?

Do you think your work has anything to do with it?

Have you given any thought to what caused the eruption? Why?

Do you feel that nervous strain mental upsets or worry has influenced the eruption?

Are you being treated for nerves, heart kidney high blood pressure or other conditions?

Do you have any allergies of which you are aware?

Why are you anxious to have this eruption treated?

If you have been to other doctors for this condition why were you dissatisfied?

Is the rash aggravated by scratching, heat, sweating clothing cold bathing, salves or lotions?

Which of these symptoms do you complain of itching burning crawling sensation, soreness, pain, tightness.

Before ending the interview the patient must be convinced that a proper diagnosis has been made and that his full co-operation as regards therapy will be forthcoming.

Special History A specialized history naturally is required in cases of occupational and contact dermatitis, drug eruptions, neoplasms, purpura, psychosomatic disease, allergic conditions, lymphoblastomas syphilis and tropical diseases.

ETIOLOGIC FACTORS

Skin conditions are produced by (1) predisposing or contributing factors and (2) precipitating, exciting or immediate causes. In some cases no sharp line exists between the predisposing and the exciting factors.

PREDISPOSING CAUSES

Heredity Most hereditary dermatoses are incurable. The probability of transmission to the offspring depends upon the type of inheritance involved. The dominant dermatoses are keratosis palmaris et plantaris, epidermolysis bullosa and ichthyosis. The recessive genodermatoses include xeroderma pigmentosum, hydroa aestivale, ectodermal dysplasias and hair and nail dystrophies.

Race Basal-cell epithelioma, psoriasis and leukoplakia are

rare in the Negro while keloids, tinea capitis, sarcoidosis, pigmentary disorders, annular syphilides, elephantiasis, superficial folliculitis and acne keloid are common in this race. Scandinavians are predisposed to tuberculosis of the skin.

Trauma may predispose to varicose ulcers, gummata, impetigo and eczematoid dermatitis. Scratching and rubbing are associated with neurotic excoriations and localized neurodermatitis. Continuous pressure trauma usually causes atrophy while intermittent pressure causes local hyperkeratosis.

Hyperhidrosis is a factor in many cases of miliaria pompholyx, dyshidrotic dermatitis and fungus infections.

Climate and Season The winter dermatoses consist of chilblains, frostbite and winter eczema. Psoriasis, seborrheic dermatitis, ichthyosis, vasospastic dermatoses, atopic eczema, xeroderma and most cases of acne are aggravated by cold. Herpes zoster and contact dermatoses due to soap, wool and furs are common in the winter.

Dermatologic disorders more frequent during the summer months include plant dermatitis, hyperhidrosis, impetigo, insect bites, urticaria, dermatomycoses, miliaria and sunburn.

Tropical disease is defined by Simons as one which, by virtue of its etiology, occurs, either exclusively or predominately in some tropical or subtropical region, where though not strictly endemic, it is either autochthonous or in residence since it has been repulsed in other areas by hygienic measures.

Physiologic States. MENOPAUSE. As a result of atrophy of the thyroid and increased compensatory functions of the pituitary and the adrenals, atrophic and neurocirculatory dermatoses are not infrequent. Kraurosis vulvae, warty nevi, hypertrichosis, localized neurodermatitis, alopecia and pruritus vulvae et al. are associated with this state.

MENSTRUATION may be associated with herpes simplex, urticaria, hyperhidrosis, acneiform lesions on the chin, and stomatitis dysmenorrhoea as a result of histaminelike substances in the circulating blood. Menstruation aggravates local pruritus from any cause and often causes a temporary flare-up of pompholyx, urticaria and generalized and localized neurodermatitis.

PREGNANCY The pregnant woman is not especially susceptible to skin disorders. Some conditions may be precipitated by pregnancy, some aggravated and a few improved by pregnancy. The only dermatosis that is directly associated with the pregnant state

is herpes gestationis, which may result in fetal death but is not serious for the mother. Acute and subacute lupus erythematosus often are aggravated and termination of the pregnancy is advisable after consultation with another dermatologist. Changes definitely associated with the condition are (1) hyperpigmentation (chloasma) (2) striae gravidarum, (3) an increase in fat tissue about the hips, the thighs and the breasts and (4) changes in the physiognomy of the face, probably due to salt retention.

Urticaria general or anogenital pruritus palmar erythema varicocities and proliferative gingivitis are not uncommon in pregnancy. Monilia of the vagina or the genitocrural region may result from a proliferation of monilia in the increased vaginal secretions.

The toxic conditions (urticaria general and anal and vulvar pruritus) usually appear during the last trimester and clear up at term.

When syphilis is acquired during pregnancy the chancre is apt to be very small and of short duration and the secondary eruption so mild as frequently to be unnoticed.

Pigmented nevi and angiomas may start to grow during pregnancy. A regrowth of hair in alopecia areata totalis may occur only to fall out after delivery. About one half the cases of psoriasis and acne improve under the influence of pregnancy.*

PURPURA is the physiologic state when acne seborrhea hyperhidrosis and certain forms of nevus tardus appear.

AGE. Infants are subject to congenital nevi infantile dermatitis and napkin eruptions. Acne, seborrhea warts and hyperhidrosis occur in adolescents. Diseases of middle age include rosacea and lupus erythematosus. Aged individuals are subject to epithelioma, keratoses (seborrheic and senile) stasis eczema, alopecia, exfoliative dermatitis, precancerous lesions and xeroderma.

SEX. Women are more subject to lupus erythematosus than men. The leukomelanoderma of syphilis is practically limited to women. Contact dermatitis from cosmetics and detergents is more frequent in the female sex, while occupational dermatitis and leukoplakia are more common in the male sex.

Environment. In clinic practice pediculosis corporis is common in the homeless vagabond and in the aged hermit. The in-

*For a good review of the subject, see Crawford, G. M. and Leeper, R. W., Diseases of the skin in pregnancy Arch. Dermat. & Syph., 61:753, 1950.

fluence of occupation in the development of occupational dermatoses is self-evident.

Recent residence in areas where specific diseases are encountered may cause the examiner to suspect such conditions as coccidioidomycosis (central California, Arizona) leprosy (West Indies, Mexico etc.), creeping eruption (Florida and Gulf beaches in the United States)

EXCITING CAUSES

Animal parasites include those which (1) suck blood but inject no foreign substance (pediculosis capitis, corporis and pubis) (2) those which suck blood and inject formic acid (*Cimex lectularis* *Pulex irritans* and *Apis*) (3) those which suck blood and insert their head into the skin (chigger and the grain-itch mite) and (4) those which penetrate the skin and burrow in it (*Acarus scabiei*) and *Ancylostoma braziliense* (creeping eruption)

Vegetable parasites (fungi or yeasts) are the cause of numerous dermatoses. In some of the diseases produced by yeasts, there is a tendency to invade the body (e.g., blastomycosis)

DISEASES CAUSED BY FUNGI include microsporiasis of the scalp trichophytosis corporis, tinea cruris, epidermophytosis, onychomycosis, favus, tinea versicolor erythrasma.

DISEASES CAUSED BY YEASTS include torulosis blastomycosis coccidioid granuloma, histoplasmosis, chromoblastomycosis, sporotrichosis, actinomycosis, mycetoma monilliasis.

Virus diseases include herpes zoster simplex and progenitalis, lymphogranuloma venereum and molluscum contagiosum

Protozoan diseases include syphilis, yaws, Vincent's angina, rat bite fever and amebiasis.

Bacterial diseases may be caused by the *Staphylococcus aureus* (furunculosis, pyoderma sycosis vulgaris and pyogenic paronychia) the *Streptococcus* (impetigo contagiosa ecthyma erysipelas) the acne bacillus (acne) the *Bacillus dyscrey* (chancre) the tubercle bacillus (lupus vulgaris, scrofuloderma, tuberculosis verrucosa cutis, etc.) Other diseases due to bacteria include anthrax tularemia glanders, erysipeloid diphtheria, rhinoscleroma and leprosy

BACTERIAL TOXINS may produce urticaria erythema multiforme erythema nodosum, pustular bacterids.

Physical irritants include wind, cold heat and sunlight.

Erythema ab igne is caused by long-continued exposure to the heat of a fireplace, a radiator or a hot water bottle. Sunlight may produce sunburn leukoderma, acute lupus erythematosus, herpes simplex, lentigo urticaria or xeroderma pigmentosum in susceptible individuals.

RADIANT ENERGY may produce x ray or radium dermatitis.

Chemical irritants vary from soap and water to complex coal-tar derivatives.

Hypersensitivity comprises a group of phenomena characterized by an abnormal, local or general, response to an ordinarily nonreacting substance. Many of these reactions are the result of a sudden endogenous ACTH stimulation of the adrenal glands (alarm reaction). If the etiologic factor continues to overwhelm the defensive forces, adrenal exhaustion follows, which may be overcome temporarily with ACTH or cortisone.

Local anaphylaxis denotes circumscribed reactions at the site of an injected antigen in allergized individuals. This type of response includes (1) the positive skin test (pollen food epidermal, fungus or bacterial) (2) the Arthus phenomenon a severe local reaction in the skin at the site of repeated injections of a foreign serum and (3) the Schwartzman phenomenon, a hemorrhagic necrotic reaction in the skin in patients with a bacterial or a fungus focus receiving an injection of an antigen with the production of an antigen antibody reaction in situ.

Sensitive State. Some patients are born with a capacity to react to various chemical and biologic agents. This "allergic" base may broaden with advancing years but may suddenly disappear at any time.

Allergy consists of atopic and nonatopic types. This mechanism is a specific altered state produced by previous exposure and made manifest immediately or shortly thereafter by later exposures to the same or a closely related substance.

ATOPIC ALLERGY is influenced by inheritance and may be characterized by a family history of asthma, hay fever migraine or urticaria, a history of infantile eczema eosinophilia, a hyper-sensitive skin and a variable psychosomatic pattern.

Atopic allergy may be provoked by exposure to one or more of the following allergens or atopens (1) animal epidermal substances (wool, silk, horse dander etc.) (2) animal and vegetable parasites (3) drugs (4) foods (5) pollen (6) protein substances (7) serum and vaccines. Atopic eczema urticaria, drug

eruptions of the hypersensitivity type and the allergic type of contact dermatitis are the more common allergic dermatoses.

NONATOPIC ALLERGY *Contact allergy* is a form of localized allergy with an eczematous reaction. It may occur in an individual at the site of exposure after an interval of time following the sensitizing contact.

Causes of contact dermatitis include the following animal, vegetable and mineral substances—soaps, dyes, medicinal drugs, anesthetics plants, clothing detergents, solvents, paints and varnishes, etc.

Drug allergy is a form of nonatopic allergy produced in susceptible individuals by the injection the ingestion the inhalation or the absorption of a drug. The clinical manifestations of drug allergy are different from those produced by the pharmacologic action of the drug. Drug eruptions are fairly characteristic for the specific drug. The drugs which frequently produce eruptions include iodides, bromides, arsenicals, barbiturates, gold penicillin and the sulfonamides.

Bacterial allergy (bacterial mycotic, virus, toxic, etc.) is that form of nonatopic allergy which is brought about by infection or by adequate contact with micro-organisms and their products. The altered reaction may consist of *hypoergy* *anergy* (immunity) or *hyperergy*. The clinical lesions include bacterids, erythema nodosum purpura sarcoid or erythema multiforme. The following is a list of agents which may cause infectious allergy:

- 1 Bacteria the organisms of tuberculosis, leprosy and soft chancre the streptococcus and the staphylococcus are also in this group.

- 2 Fungi the organism of trichophytosis, *oidiomycois* *sporotrichosis* and *blastomycois*.

- 3 Viruses variola, herpes and lymphogranuloma venereum.

- 4 Protozoa syphilis, yaws and leishmaniasis.

Spontaneous cure in many bacterial and virus diseases by the formation of antibodies or the development of protective enzymes is known as *esophylaxis*.

Foreign-protein allergy is a form of nonatopic allergy which is produced by adequate exposure to foreign substances of a protein nature. The reaction, which consists of an immediate or delayed reaction anaphylactoid shock and urticaria usually is manifested by subsequent exposure to the specific allergen or immunologically related substances.

Physical allergy is a nonantigenic and nonatopic type of abnormal reaction in susceptible individuals, produced by exposure to heat, cold, light and mechanical irritation in an amount and a manner usually harmless to normal persons. These physical agents probably produce a histaminelike substance in the skin which acts upon the skin capillaries altering their permeability resulting in pruritus or urticaria.

Neoplastic diseases include (1) malignant growths (carcinoma, sarcoma and melanoma) and (2) the benign tumors, (lipoma fibroma, sebaceous cysts, cutaneous horns, dermoid cysts, paraffinoma, etc.)

Circulatory disturbances may produce Buerger's disease gangrene, gravitational eczema and Raynaud's disease.

Psychosomatic dermatoses may be the result of vasomotor pathology (urticaria, rosacea, dermatographism) nerve innervation pathology (hyperhidrosis, pruritus, acarophobia) increased tissue irritability (atopic eczema) psychogenic reaction (trichotillomania, factitious eruptions, neurotic excoriations)

Unknown Etiology These disorders include pityriasis rosea psoriasis, lichen planus, lupus erythematosus, lymphoblastomas, pemphigus mycosis fungoides and vitiligo. Until a proved etiologic factor is discovered, we must continue to employ the unsatisfactory term "individual susceptibility."

CUTANEOUS DIAGNOSIS

After the student acquires a general knowledge of the causes of skin diseases he will find that diagnosis will be simplified if he studies each case in six consecutive steps (1) general inspection of the entire eruption (2) regional localization or distribution (3) determination of the primary lesion (4) determination of the secondary lesions (5) gross pathologic changes (6) palpation.

A correlation of history examination biopsy and laboratory studies is then necessary for the final interpretation in all but the obvious cases of skin disorders. In some cases an unequivocal diagnosis cannot be made for several days because (1) clinical lesions are atypical (2) previous treatment has blurred eruption (3) histologic sections are not typical (4) history is not reliable and (5) laboratory data is inconclusive.

INSPECTION

General Inspection. The first step in the diagnosis of an eruption or a cutaneous lesion is a general inspection of the disorder. This includes observation of the primary and the secondary lesions, evidence of previous therapy, extent of involvement, presence of secondary infection, grouping, configuration, color and surface. The eruption may be uniform or polymorphous, symmetrical or asymmetrical or it may occur in different stages of evolution and involution. The lesions may be solitary, few in number or numerous. In arrangement, the eruption may follow no definite pattern (psoriasis). It may occur along the lines of cleavage (pityriasis rosea) or along the distribution of the cutaneous sensory nerve trunks (herpes zoster) or it may tend toward grouping (syphilis). The eruption may consist of lesions which take the form of rings (erythema multiforme), crescents (measles) or bizarre plaques (mycosis fungoides).

MORPHOLOGIC STUDY. The morphology of a lesion is dependent on various factors including soil, anatomic site, selective tissue reaction, number and virulence of the organisms, local and general immunity, duration of the disease and the speed and the intensity of the defense mechanism. Thus, in certain forms of cutaneous tuberculosis the skin is anergic; in acute dermatophytosis the skin is often allergic. Kerion and gummas represent a state of localized allergy. The roseola of secondary syphilis is the result of a specific generalized allergy in which the organisms are killed *in situ*.

Ringed eruptions are produced by a central immune zone, a cessation of growth or a peripheral multiplication of the organisms. The spreading of a chancroid is caused by the absence of a marginal zone of immunity. Satellite lesions are produced by the presence of a partial zone of immunity.

Polymorphism. Various regions of the skin may react differently to the same cause depending on the nerve and the blood supply, the presence or the absence of hair, sebaceous and sweat glands and pigmentation and whether the exposed or the covered parts of the body are affected. For example, an infection like monilliasis may produce a scaly eruption on the face, a curdy white patch on the buccal mucosa, a fissured lesion at the commissure of the mouth and eczematoid lesions on the hands or the feet. Drug eruptions are sometimes macular or urticarial on the trunk and purpuric on the legs.

Routine of Inspection. Natural light is the best for observing dermatologic conditions. Regarding the patient's complaint, he should be entirely exposed and the entire cutaneous surface and the mucous membranes should be examined in an orderly routine. Starting with the head, the following should be examined in regular order: face, eyes, ears, neck, axillae, upper extremities, fingers, arms and forearms, trunk, groins, genitalia, anal region, buttocks, lower extremities and palms and soles. Hair, the nails and the teeth should be examined next. Then follows an examination of the mouth, including the tongue and the tonsils, and the genitalia if indicated. The presence of localized or generalized adenopathy may be important. A small hand lens or magnifying glass is useful for the study of individual lesions.

While examining the skin, it is important to note the condition of the underwear, the soiled dressings, the presence of antiseptic dyes used, the trusses and the style of hair, all of which are clues. At the same time the presence of fear, anxiety, and distrust must be noted.

Special attention should be given to the favoring of the use of clean sheets or towels and avoidance of unnecessary exposure.

REGIONAL LOCALIZATION

The second step in the diagnosis of an eruption is the regional localization. Practically every disease in its typical state has a more or less selective affinity for certain areas. These must be memorized if skill in cutaneous diagnosis is to be acquired. The affinity of diseases for certain sites is determined by accidental, biologic, etiologic and anatomic factors.

Localization of Some Common Dermatoses

Chronic psoriasis—extensor surfaces of elbows, knees, legs, humbo-sacral area, scalp.

Pityriasis rosea—along the lines of cleavage of the trunk, sides of the neck, upper back.

Scabies—lower buttocks, penis, interdigital spaces, abdomen, anterior wrists.

Acne—face, upper back, shoulders and mid-thighs.

Seborrheic dermatitis—scalp, retroaural areas, eyebrows, mid-chest, upper back, umbilicus.

Herpes zoster—usually on one side of the trunk from the mid-back to the mid-abdomen.

Atopic eczema—antecubital and popliteal spaces, face anterior neck.

Acute neurodermatitis—eyelids antecubital spaces, "V" of neck.

Tinea versicolor—chest upper back, mid-abdomen.

Erythema multiforme—extensor surfaces forearms, dorsal surfaces of hands, face mouth.

Contact dermatitis—at site of maximum exposure to suspected contactant with spreading to contiguous areas.

DETERMINATION OF THE PRIMARY LESION

The detection of the primary original or initial lesion in the presence of secondary irritation or "blurring" of the eruption often helps to classify the disease and to establish the correct diagnosis. These important lesions include the following

Macules (spots) are flat circumscribed discolorations of the skin. Freckles, purpura and vitiligo are familiar examples of macules.

Wheals (welts) are raised edematous macules transitory in character pink in color and associated with pruritus. Urticaria is an eruption which consists entirely of wheals.

Papules (pimples) are circumscribed elevated lesions resulting from an infiltration in the corium. They may enlarge peripherally to form a plaque. The surface of the lesion may be conical rounded or flat the shape round, oval or rhomboidal the color red pink violaceous, yellow brown or black.

Nodules (tubercles) are large papules resulting from an infiltration in the deep corium. Soft nodules occur in von Recklinghausen's disease the gumma of syphilis and the nodules of leprosy are examples of hard nodules. In lupus vulgaris, late syphilis of the skin and basal-cell carcinoma nodules often are found in the vicinity of scar tissue.

Tumors are large nodules or solid elevations. Pedunculated tumors have a constricted base sessile tumors have a flat base. The neoplastic tumors include carcinoma, sarcoma and melanoma. The benign tumors include xanthoma, gummata, lipomata and fibromata.

Vesicles (small blisters) are pea sized collections of serous fluid resulting from a circumscribed edema in the epidermis. The roof of a vesicle usually consists of the stratum corneum. Acute dermatitis of external origin usually consists of vesicles and erythema.

Bullae (blebs) are thumb-nail-sized collections of serous fluid. They may be flaccid or tense. The contents consist of serum which may be hemorrhagic or purulent. Bullae may be caused by local irritants (phenol) drug allergy (iodides) vascular disturbances (Raynaud's disease) physical trauma (frostbite) congenital tissue defects (epidermolysis bullosa) and streptococcal infections (Impetigo)

Pustules result from secondary infection of papules, vesicles or bullae. They are usually yellow or greenish in color. Large pustules result in abscess formation. The position of the pustule may determine the character of the clinical lesion. In *syccosis vulgaris* the mouth of the pilosebaceous follicle is involved. In *acne*, the upper third and in *furuncle*, the lower third of the follicle.

Barrows are macroscopic slightly elevated grayish dotted, tortuous or straight lines found especially on the wrists and the outer edges of the hands in scabies.

Cysts are noninflammatory collections of fluid or semisolid substances, surrounded by a well-defined wall.

Comedones (blackheads) are white, gray or black noninflammatory plugs in the pilosebaceous openings, consisting of dried sebum epithelial debris and bacteria. They are always present in *acne vulgaris* and acneiform eruptions resulting from contact with oils, grease and chlorine compounds and also may occur in seboreic and senile skins.

DETERMINATION OF SECONDARY LESIONS

Scales are the result of imperfect cornification. The formation of scales over large areas is called exfoliation or desquamation. Scales may be branny or shiny thin or thick, white, yellow or gray limited to the surface of a papule (*pityriasis rubra pilaris*) or occurring in large sheets (*scarlatiniform erythema*)

Excoriations (abrasions erosions or scratch marks) are linear or punctate breaks in the continuity of the skin caused by voluntary or involuntary trauma. They are seen in such pruritic dermatoses as *pediculosis capitis* and *corporis* atopic eczema and neurotic excoriations. The regional lymph nodes often are palpable. Excoriations over large pruritic areas result in linear scratch marks. They heal without scarring.

Fissures (cracks) are breaks in the epidermis down to the level of the dermis or the mucous membranes due to movement.

They often occur on lips, finger tips, retroaural folds, creases of the palms and soles, intergluteal cleft and under the breasts. Fissures often are the portal of entry for bacterial invasion. Chapping and extreme dryness from any cause produce fissures.

Crusts (scabs) are thin or thick, yellow brown or green masses of dried serum, pus or blood. Thick crusts usually are associated with ulcerative conditions. Thin orange-colored crusts are found in impetigo. Weeping or oozing eczema is associated with a brittle glazed honey-colored crust composed of serum from ruptured vesicles which adheres to dressings. Crusting in eczematous dermatitis suggests an acute process. The odor of a crust may be diagnostic: the mouse odor of favus, the musty odor of pemphigus, the putrefactive odor of gangrene and advanced carcinoma and the foul odor of an ulcerating late syphilide.

Ulcers are circumscribed areas of tissue destruction. Superficial ulcers are limited to the epidermis, as in impetigo contagiosa. Carcinoma, syphilis and tuberculosis may produce deep ulcerations which involve the corium and the subcutaneous tissues. Ulcers may result from local infection, nerve and vascular disturbances or from metabolic disorders (sickle-cell anemia).

Scars (cicatrices) result from inflammatory or traumatic destruction of the elastic and fibrous tissue in the corium and deeper parts. Their size, shape and configuration are sometimes suggestive of the character of the original disease.

Will it leave a scar? is a question often asked by patients who consult the physician for the removal of a nevus or skin neoplasm. The size of the scar will depend on the skill employed in its removal but a hypertrophic scar or even a keloid may develop in spite of the best of care.

A complete history should be taken when nontraumatic scars are discovered in the routine examination.

Pigmentation is due to a deposit of melanin, blood pigment or foreign substances in the basal-cell layer of the epidermis or deeper tissues.

GROSS PATHOLOGIC CHANGES

Atrophy may be primary or secondary depending on the presence or the absence of pre-existing inflammation. The skin is thin, wrinkled, transparent and inelastic with loss of hair follicles, sweat and sebaceous glands. Scales, pigmentation, keratoses

and dilated capillaries or venules may be present or absent in atrophic areas.

Crusting is the presence of dried serum, pus or blood on a weeping surface. Crusts may be adherent or easily removed.

Cyanosis is a bluish vascular discoloration of the skin usually affecting the extremities and often associated with a low vascular tone and hyperhidrosis.

Depigmentation consists of white areas resulting from loss of pigment (melanin) which may be congenital (albinism) or acquired (vitiligo) localized or generalized.

Edema of the skin may be inflammatory (erysipelas) or non-inflammatory. Angioneurotic edema is a sharply circumscribed edema in the subcutaneous tissues. Acute dermatitis of the legs and face is usually associated with edema.

Echymoses are large extravasations of blood resulting from rupture of the skin capillaries. They do not disappear on pressure.

Exzematization is a condition produced by irritating applications, trauma, infection or autosensitization in which exzematoid changes appear on a normal skin or in previously dry dermatoses.

Erythema is the commonest and earliest sign of cutaneous pathology. It is due to a dilatation of the arterioles and the capillaries. Erythema may be punctate, patchy or diffuse. It is difficult to detect this condition on the skin of the Negro.

Hyperemia. Active hyperemia is characterized by an increase of arterial blood as in erysipelas. Passive or venous hyperemia is the result of a retarded outflow of blood as in acrocyanosis.

Hypertrophy of the skin may involve all the layers of the skin (verruca vulgaris) or it may be limited to a single layer (clavus or callus).

Keratosis are circumscribed hyperplasias of the horny layer (stratum corneum) resulting from chemical, inflammatory or actinic causes. In some cases, they are subject to malignant degeneration. Senile keratosis are the result of degenerative changes in the skin. The seborrheic type has been considered as a delayed form of nevus. Arsenical keratosis result from a specific type of local epidermal stimulation.

Lichenification is a change in the skin characterized by thickening, pigmentation and an exaggeration of the normal creases. This condition is the result of constant rubbing of a localized patch of pruritus.

Telangiectasia are clusters of fine dilated capillaries. They usually are found on the face but may occur anywhere. The condition occurs in lupus erythematosus, chronic radiodermatitis, rosacea and other conditions.

Vegetations are vascular cauliflowerlike masses localized to moist areas (axillae, groins and anogenital region). They are produced by a hypertrophy of the prickle-cell layer of the skin.

CONDYLOMATA LATA are vegetative hypertrophies resulting from syphilitic infection.

PALPATION

The presence of infiltration in a lesion is often a valuable diagnostic sign. This is felt with the middle finger or observed with the use of a glass slide or a diascop. There is an absence of infiltration in all superficial inflammatory diseases. The infective granulomas especially syphilis, tuberculosis and carcinoma, are characterized by more or less deep infiltration. The apple-jelly nodules of lupus vulgaris are easily recognized by the use of a diascop.

SUBJECTIVE SYMPTOMS

Subjective symptoms are manifested by sensations and vary according to the nature of the disease, the anatomic site and the psychic make up of the individual. The symptoms of emotional hypersensitivity include itching, burning, tingling, smarting, biting and crawling sensations and pain. The symptoms of paresthesia are numbness, pressure, hot and cold sensations and a feeling of constriction. Localized anesthesia, or absence of sensation may occur in leprosy, factitious dermatitis, syringomyelia or tabes dorsalis.

Itching may be absent, mild, moderate, severe or intolerable. It may be intermittent or continuous, present during the night and absent during the day. If there is no apparent cause for the itching and if anesthesia of the palate and of the sclerae is present, the condition is probably psychosomatic. The presence of pruritus can be determined easily by observing the following signs: scratch marks, excoriations, lichenification, small blood crusts and, in old cases, pigmentation and linear scars (e.g. pediculosis corporis.)

The dermatoses characterized by severe itching include scabies, pediculosis, urticaria, dermatitis venenata, lichen planus, dermatitis herpetiformis, neurotic excoriations, atopic eczema and pruritus ani et vulvae.

Pain occurs in furuncles, abscesses glomus tumor herpes zoster, neuroma, kelomyoma and fissures.

DIAGNOSTIC METHODS

WOOD'S LIGHT

Wood's light consists of wavelengths of 3650Å produced by filtering ultraviolet rays through nickel-oxide glass. Apparatus in which the filter is in the glass surrounding the filament is available from several manufacturers. This light causes organic material to fluoresce.* This property fluorescence is the absorption and the conversion of ultraviolet rays from the Wood light into rays of a longer wavelength which are visible as long as the source continues. It is used primarily in the diagnosis of ringworm of the scalp, to observe the extent of the infection in the detection of carriers, and to detect relapses after apparent cure also in the diagnosis of tinea versicolor and the study of ringworm colonies to determine the extent of leukodermic areas, to detect porphyric urine to verify the diagnosis of molluscum contagiosum, and to reveal the subclinical macular lesions in seronegative syphilis. Ronchese has demonstrated a vivid orange reddish fluorescence in the necrotic ulcerated surfaces of squamous cell carcinoma in contrast with the uniform deep violet discoloration of other ulcerated lesions.

BIOLOGIC TESTS

Biologic tests are sometimes necessary in the diagnosis of cutaneous disorders.

The basal metabolism test is of value in evaluating the thyroid factor in ichthyosis, general pruritus, myxedema, acne vulgaris and rosacea. More accurate than the B.M.R. is the isotope iodine 131 test. The amount of the uptake of the orally administered drug compared with the amount of the tracer excreted in the urine is measured.

Urinalysis. The urine should be examined routinely in extensive eczemas, pyogenic disease cases in which glycosuria is suspected drug eruptions and febrile conditions. During ACTH and cortisone vitamin D₂ and gold therapy urinary studies are important to detect disturbances in the excretory functions of the

Costello, M. J. and Lottenberger, L. V. Fluorescence with the Wood Light as an Aid in Dermatologic Diagnosis, N. Y. State J. Med. 44: 1774, 1944

kidneys. Often melanin is found in late cases of malignant melanoma, and porphyrins are found in acute porphyria.

In allergic conditions, the diagnosis is confirmed but not made with patch scratch or intradermal tests, and the Prausnitz Kùstner method of passive transfer.

Skin testing with food allergens as a diagnostic aid has been a complete failure in my experience. We agree with Bruce Pearson's statement that skin tests do not provide a short cut to a speedy diagnosis and in the hands of those who expect too much, they often are misleading.

Hematologic studies are important in the lymphoblastomas, drug eruptions, the deep fungus diseases, acute lupus erythematosus, purpuras, allergic eczemas, diseases treated with ACTH heavy metals and leukocytic nuclear toxins.

Blood chemistry studies are important in all the metabolic disorders, including xanthoma calcinosi cutis, hyperglycemic furunculosis senile eczema and scleroderma.

"L.E. cell" blood studies are made in suspected cases of acute lupus erythematosus during the active stage.

Serum protein studies are important in chronic senile eczemas, leg ulcers and pemphigus to detect hypoproteinemias. The albumin-globulin ratio may be reversed in acute lupus erythematosus, sarcoidosis and lymphogranuloma venereum.

Vitamin A serum levels and absorption curves are important aids in the diagnosis of follicular and hyperkeratotic dermatoses resulting from vitamin A deficiencies.

Bone Marrow Studies Examination of the sternal marrow may confirm or establish the diagnosis in acute lupus erythematosus, chronic leukemias, Hodgkin's disease metastatic carcinoma and cases of chronic exfoliative dermatitis of unexplained etiology (Pascher *et al*).

The ultraviolet sensitivity test is used in the diagnosis of the photosensitivity dermatoses. The back or the chest is covered with a black cloth containing holes the size of a 5-cent piece. These openings are then exposed to the rays of an ultraviolet light lamp at a distance of 30 in. for periods of 10 15 and 20 seconds each. Then the results can be compared with exposures on a normal individual for a control.

INTRADERMAL TESTS

Tuberculin tests are made with solutions of old tuberculin (1:100 to 1:1,000,000).

GRADED TUBERCULIN TESTS For routine testing, the tuberculin patch test or a 1:10,000 dilution is used with a control.

If there is reason to suspect a visceral focus, a 1:1,000,000 dilution should be employed to avoid a flare-up. The degree of hypersensitivity is determined by the use of several graded dilutions ranging from 1:1,000,000 to 1:1,000. False negative reactions may be due to impotent tuberculin, subcutaneous instead of intradermal injection, a state of anergy or the presence of a bovine instead of a human infection.

Trichophytin and oldiomycin tests for fungus and yeast diseases are unsatisfactory because of the frequency of non-specific reactions, and the long duration of sensitivity to previous infections, resulting in many false positive readings.

Dimelcos and Ito-Reenatierna intradermal tests for chancreoid are made with a pure culture of streptobacilli. The reading is evaluated after 48 hours.

Chick Embryo Antigen (Lygranum) Test for Lymphogranuloma venereum. In this modification of the original Frei test, 0.1 cc. of the commercial antigen is injected intradermally on the flexure surface of the forearm, using a control. The test is read after from 48 to 96 hours.

The Kveim test is a cutaneous test used in the study of sarcoidosis, although there is no agreement as to its specificity. One tenth cubic centimeter of antigen prepared from lymph nodes (or cutaneous infiltrates or tonsils) of patients with sarcoidosis is injected intracutaneously usually on the flexor surface of the forearm. A positive reaction consists of an infiltrated area, papule, nodule or superficial necrosis with ulceration, which appears at the site of injection sometimes within two weeks, sometimes not for several months, and it may be observable for many months thereafter. In doubtful cases, a histologic examination of the papule with demonstration of a tubercloid structure would tend to support the diagnosis of sarcoidosis.

ROENTGENOGRAPHIC STUDIES

Involvement of the bones and the viscera may occur in various cutaneous diseases and may be of diagnostic or prognostic value.

X ray studies of the bones are indicated in congenital syphilis, gummata, sarcoidosis, neurofibromatosis, Hand-Christian disease, porphyria, urticaria pigmentosa, cavernous and extensive port wine angiomas and in the metastatic type of skin malignancies.

Roentgenograms should be taken of the spine in bilateral herpes zoster to eliminate carcinoma or tuberculosis.

X-ray studies of the lungs are indicated in sarcoidosis, skin tuberculosis, blastomycosis, coccidiomycosis, in indolent skin disorders associated with malnutrition, chronic nail disorders and as a preliminary to steroid therapy.

MICROSCOPIC EXAMINATIONS

Smears are made routinely for Vincent's disease, staphylococci and streptococci conditions, leprosy and secondarily infected lesions.

Darkfield Examination for *T. pallidum*

Cultures are employed in the diagnosis of fungus and yeast infections. Sabouraud's media are used universally. Care must be taken in the selection of material. The stumps of the hairs are used in tinea capitis; the hairs in the pustules or the nodules in tinea sycosis; the roofs of the vesicles and the margins of the patches, in tinea cruris, tinea circinata and dermatophytosis of the hands and the feet. Hanging-drop preparations are used in the detailed study of cultural growths.

Direct Examinations of Scrapings for Fungi. After the site has been selected it should be scrubbed with 70 per cent alcohol, and a generous portion of actual scales, not pus, serum or crusts, scraped off with a dull sterile knife. The material for study is divided into two portions, one of which is placed between two sterile glass slides for cultural study. The other portion is placed on a slide, a few drops of ether are added and it is permitted to evaporate. Then 1 or 2 drops of 40 per cent potassium hydroxide or lacto-phenol cotton blue is placed on the material, and a cover slip is added. The preparation is warmed gently for a minute, allowed to stand for from 10 to 15 minutes and then examined. Permanent preparations can be obtained by replacing the potassium hydroxide with glycerin.

Spinal fluid examinations are performed in syphilis to determine the presence of the disease in the central nervous system and in torulosis and other deep fungus infections to detect meningeal involvement. Lumbar puncture has been used in the treatment of idiopathic severe pruritus and resistant lichen planus.

CAPILLARY MICROSCOPY

Capillary microscopy is employed in the study of the superficial blood vessels under direct illumination after the skin has been

made transparent by means of a drop of oil applied to the field to be examined. The method is used by research workers in the study of the anatomy the physiology and the nature of vascular and allied dermatoses.

BLOOD SEROLOGIC TESTS FOR SYPHILIS

For a detailed discussion of the Wassermann the Kahn, the Kline and other standard tests, the reader is referred to Todd, J. C., Sanford A. H., and Wells B. B. *Clinical Diagnosis by Laboratory Methods*, ed. 12 Philadelphia, Saunders 1953

CYTOLOGIC STUDIES

The Tzanck test is used as an aid in the diagnosis of pemphigus. It is a method of cytologic diagnosis, but because the prognosis is so grave we still must depend on clinical features and the course of this disease. An early clear young vesicle is punctured with a sterile scalpel, the serum wiped away and the base of the lesion gently scraped. Then the material is fixed in methyl alcohol and stained with the Giemsa stain. The pemphigus cell is loose and detached its nucleus is large in relation to the cytoplasm which is deeply stained especially at the periphery and the nucleus is pyknotic (acantholysis).

The technic also is used as a rapid means of differential diagnosis in vesicular and bullous dermatoses when the usual biopsy would be time-consuming or disfiguring. It is a speedy simple, inexpensive, painless means of diagnosis, but, of course only the expert should perform this test.

The cantharides blister test (Brennan) also is used in cytologic diagnosis. A portion of cantharides cerate (4x4x2 cm.) is applied to an area of apparently normal skin for 6 hours and the resulting blister scraped and stained as in the Tzanck test.

BIOPSY

Histologic examination is important when an accurate clinical diagnosis cannot be made, to corroborate the diagnosis in lymphoblastomas when the blood picture is atypical in suspected syphilitic lesions when the serology is negative and for purposes of investigation. A discrepancy between the clinical and the pathologic diagnosis may be the result of faulty selection of the site for biopsy. Such diseases as pityriasis rosea, scabies, herpes zoster, variola and varicella are diagnosed more readily by clinical observation. There is little danger of removing tissue for biopsy

except in melanomas, Paget's disease and acute suppurative conditions.

The histologic examination should be made by a dermatopathologist who is less likely to render a report of "chronic inflammatory tissue." A fully developed lesion and the advancing border of a tuberculous, syphilitic or malignant lesion should be selected for the site of the biopsy. The center of an ulcer irradiated lesions lesions undergoing involution or secondarily infected ones should not be used.

Excision of skin lesions with the knife is preferred to removal by the cautery current or cutaneous punch. Although an oval excision is best for cosmetic reasons a square piece of tissue produces less waste. Paraffin serial sections are more satisfactory than frozen sections because they can be cut thinner to permit study of the minute structures in the skin. The section should be fixed in alcohol for routine study but if fat stains are to be used one half of the tissue should be fixed in 10 per cent formalin solution. Vascular tumors should be excised with the knife blade of an electrocautery mucous membrane lesions with the punch biopsy knife material from sinuses, with the curet, and material from cysts, by paracentesis.

The clinician should furnish the pathologist with all the diagnostic possibilities in order to aid him in arriving at a definite conclusion.

Precautions to assure a report of maximum value include (1) avoid the use of dyes when preparing site (2) section should be deep enough to include the entire thickness of the skin mucous membrane or tumor (3) an adequate border of normal tissue should be included for purposes of comparison (4) the incision should be planned to avoid or minimize disfigurement (5) if cautery must be used avoid cooking the tissue (6) do not crush specimen with forceps (7) place specimen in proper solution immediately to avoid drying out and (8) label and attach complete report to specimen.

CUTANEOUS MANIFESTATIONS OF SYSTEMIC DISEASES

A study of the skin and the mucous membranes may establish or confirm a diagnosis of internal or visceral disease.* The im-

*Wiener Kurt. The Skin Manifestations of Internal Disorders, St. Louis, Mosby, should be consulted for a more detailed discussion and review of this subject

portant cutaneous eruptions and symptoms associated with systemic disease include the following

Nevi and Nevoid Diseases. Extensive nevi of the face are sometimes associated with angiomas of the brain adenoma sebaceum, with tuberous sclerosis and von Recklinghausen's disease, with congenital visceral tumors.

Metabolic Disorders. Diabetes may result in (1) toxic symptoms (localized or generalized pruritus) (2) increased susceptibility to pyogenic infections (furunculosis, cellulitis, carbuncle) (3) increased susceptibility to parasitic dermatoses (monilliasis, dermatophytosis) (4) vascular disturbances (gangrene and necrosis) and (5) abnormal lipid metabolism (xanthoma, necrobiosis) Nutritional senile eczema the localized eruption of amyloidosis and scleroderma are also metabolic disorders. Some types of palmar erythema, pulsating spider nevi and acquired porphyria are caused by hepatic cirrhosis.

Pigmentation or discoloration is often a valuable diagnostic sign. The yellow palms of carotinemia the dusky-red face and chest of polycythemia vera and the slate-blue color in argyria are of interest.

Adrenal disturbances In Addison's disease the pigmentation of asymptomatic visceral malignancy in adults, acanthosis nigricans, the hemochromatosis of hepatic disease, the melanoderma of chronic arsenic intoxication and the generalized pigmentation of abdominal tuberculosis and melanoma are also of interest to the internist.

Vascular Diseases. The early signs of Raynaud's disease and Buerger's obliterative endarteritis are found in the skin. Acute lupus erythematosus is probably the cutaneous expression of a generalized vascular disease with endocardial and nephritic complications. Rosacea is a cutaneous reaction which is produced indirectly by various visceral disturbances. Periarteritis nodosa may present an eruption of subcutaneous nodules or purpuric lesions.

Visceral carcinoma not only may present metastatic nodules in the skin but also occasional intense pruritus, velvety pigmented plaques in the axillae or groins (acanthosis nigricans) reticulated melanosis, purpura, pemphigoid eruptions herpes zoster and dermatomyositis.

Pruritus. First we must determine whether the pruritus is psychogenic inflammatory or caused by animal parasitic invasion. This requires a complete history and examination of the entire

skin. In the absence of an eruption, this symptom never should be dismissed lightly. The type localized to the vulva or the anus sometimes is associated with menopausal changes or psychosomatic influences. Pruritus in the aged may be caused by arteriosclerosis or suburemic states. Myxedema, hyperthyroidism, diabetes and visceral malignancy sometimes are associated with severe generalized pruritus. Pruritus is often an early symptom in the lymphoblastoma group of diseases which includes leukemia (myelogenous, lymphatic and monocytic), mycosis fungoides, lymphosarcoma and Hodgkin's disease. A nonspecific or specific eruption occurs in over 10 per cent of the cases in this group.

Allergy is a factor in contact eczema, urticaria, purpura, angioneurotic edema and fungus "id" lesions.

Local Infection. The role of chronic infections in the teeth, the tonsils, the sinuses, the ears, the gallbladder, the prostate, etc., as a contributing factor in the causation of rosacea, acne vulgaris, erythema multiforme and chronic urticaria is still a controversial issue. Each case must be decided on its merits. Before removing the foci, the eruption should be well controlled by local therapy and an antibiotic given before and after the operation.

Infections of the skin with visceral involvement include tuberculosis, syphilis, blastomycosis and tularemia. Chronic ulcerative colitis may coexist with pyogenic ulcers on the skin.

Vitamin Deficiency. A deficiency of vitamin B₂ is found in pellagra; vitamin C in some purpuric eruptions; vitamin A in many dry follicular eruptions; and vitamin E in some of the collagen diseases.

Photosensitization may produce indirectly acute or chronic eruptions in individuals who are hypersensitive to light of various wavelengths. Xeroderma pigmentosum, chronic actinic dermatitis, hydroa vacciniforme, congenital and acquired porphyria, pellagra, acute lupus erythematosus and certain drug eruptions (the sulfonamides, silver) are produced in part by a sensitivity to light.

Functional states characterized by fatigue, nervous tension or emotional states may initiate urticaria, acute disseminated neurodermatitis, atopic eczema, pompholyx and hyperhidrosis.

Diseases of unknown etiology. Sarcoidosis, acute lupus erythematosus and the lymphoblastomas are often first diagnosed by the appearance of suggestive lesions on the skin.

FUNCTIONS OF THE SKIN

The main function of the skin is to act as a coat of armor or an exterior protective covering. Temperature regulation is controlled by the delicate vasomotor mechanism. The secretion of sweat plays an important part in regulating the body temperature and in the self-sterilizing function of the skin. Together with the secretion of the sebaceous glands, the skin is kept soft and pliable. The fatty acids in the scalp sebum of the adult have fungicidal properties.

Keratinization Cycle. Desquamation varies from 6 to 14 Gm. daily and requires replacement by mitoses in the Malpighian layer. The mitoses appear rhythmical, and cellular division is more active at night. Choline, found in large amounts in the scales may act as a stimulus for continued mitoses.

Autogenous Disinfection of the Skin. Certain factors may regulate the number of saprophytic and pathogenic bacteria and fungi on the skin namely desquamation of the stratum corneum desiccation presence of fatty acids in the sebum acidity of the skin surface in health and certain unknown antibiotics elaborated in the skin.

Acid Base Balance of the Skin. The average pH of the skin is about 5.4 but the figure is variable in different parts of the body. Axillary, anal, inguinal and interdigital areas usually are neutral or alkaline and thus, are more predisposed to bacterial infection.

Sex Hormones. Estrogen and androgen are present in the skin in varying amounts depending on the age of the patient and the state of the adrenals, the pituitary, the ovaries and the testes. Tissue sensitivity to these hormones varies. Apparently there is a definite ratio of androgen to estrogen to maintain normal skin health.

Enzyme Systems. The formation of mucin, melanin and hyaluronic acid probably is activated by an enzyme system.

Peripheral Blood Flow. The capillaries of the skin maintain nutrition help to regulate body temperature and supply turgor to the skin.

Water Metabolism. There is a constant interchange of fluids and electrolytes between the blood and the corium and the subcutaneous tissues depending on the permeability of the capillary walls, the size of the capillary bed, the osmotic pressure, the con-

centration of plasma proteins and the amount of extravascular pressure.

Important metabolic processes occur in the skin including those of sulfur and cholesterol pigment formation and carbohydrate storage.

Permeability varies according to whether the skin is intact or broken. Injured skin is more likely to absorb toxic drugs. Estrogenic substances heavy metals aniline dyes and the sulfonamides, but not the antihistaminics, may be absorbed through the unbroken skin.

Aminoacids occur on the skin surface from "insensible" sweat gland activity. Individual and regional variations in the concentration of the aminoacids may be responsible for differences in susceptibility to skin infections.

Lipids The simple lipids (neutral fats) and the compound lipids (phospholipids lecithin and cephalin) on the surface of the skin are derived from the sebaceous and the sweat glands and the epidermal cells. The free fatty acids in the sebum have a fungistatic and bactericidal action. The lipids may be controlled by endocrine and nerve influences through an enzyme mechanism, but the physiology and their function is not entirely understood.

Sensory As a sense organ the skin is unique being well supplied with sensory nerves. The skin is sensitive to pain pressure touch, cold, heat and trauma. The intensity of these sensations vary in different areas the tactile sense organs being thickest on the finger tips and palms.

Cutaneous reflexes consist of the pilomotor reflex to cold (goose-flesh) the flushing reflex to anger shame or embarrassment and the scratch reflex, a protective mechanism. The sensation of itching may be caused by edema in the prickle-cell layer from various external and internal causes or from a change in surface tension in the layers of the epidermis.

ANATOMY AND PATHOLOGY OF THE SKIN

In order to evaluate properly the abnormal clinical and microscopic changes in the skin it is important to be familiar with the normal regional variations of the cutaneous pattern as well as the racial features, and temporary changes due to age physiologic causes, occupation and environment. A study of microscopic sections of the normal skin from scalp face palms, soles and genitalia etc

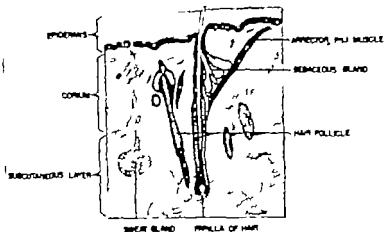


FIG. 1. Vertical section of the skin (diagrammatic) illustrating the differentiation of the skin into the epidermis, the corium and subcutaneous layers.

is of great value in order to appreciate the finer diagnostic and therapeutic difficulties in dermatology.

The skin consists of two continuous layers: the cellular epidermis, which is derived from the ectoderm, and the fibrous cutis, or corium (true skin) which originates from the mesoderm. The subcutaneous tissue or hypoderm upon which the skin rests, is composed of a loose reticulated connective tissue framework and fat cells.

The skin is thickest on the scalp, the palms and the soles and thinnest on the eyelids, the penis, the anterior neck, the flexure surfaces of the arms and the forearms, the cubital, the popliteal and the interdigital spaces. Therefore, caution must be used when prescribing topical medication, x-rays or ultraviolet radiation for these areas.

EPIDERMIS

The epidermis has no blood supply but gets its nutrition from the underlying cutis. Its cells, which are derived from the matrix, the germinal or basal-cell layer, are in a state of perpetual flux, new cells forming, and old ones being cast off as scales.

Atrophy of the epidermis may occur through mechanical or cellular pressure from within or from without. It also occurs physiologically in senile degeneration of the skin.

Hypertrophy of the epidermis is an increase in all the layers and is present in the common wart, for example. A single layer (stratum corneum) is involved in ichthyosis.

The epidermis consists of five cellular layers each representing a different stage in the evolution of a fully formed cell of the stratum corneum.

The germinal or basal-cell layer the lowermost layer is a single layer of cells possessing high vitality. They contain and produce normal pigment granules. The individual cells are separated by intercellular spaces which are spanned by thin parallel fibers, or bridges. Pathologic changes which do not extend below the epidermis are healed by a regeneration of the basal layer without scarring. The basal-cell layer shares in all proliferative processes without a break in its continuity. In malignancy however the cells of the prickle-cell layer break through it.

The prickle-cell layer (rete or stratum spinosum) consists of several layers of polyhedral and cylindrical cells. The cells of this layer are attached to one another by protoplasmic bridges, or prickles, which radiate in all directions. They supply solidity to the layer and nutrition to the cell. The spaces between the cells are connected with the lymph channels of the cutis. The layer itself forms a regular wavy outline separated from the corium by the basal-cell layer and forming rete pegs which are separated from one another by the papillae of the corium.

ACANTHOSIS. A hypertrophy of the prickle-cell is called acanthosis. It results from a stimulation of the basal-cell layer by causes within or outside the skin. Acanthosis is characterized by a perfect basal-cell layer, mitoses in the prickle cells and retention of the protoplasmic bridges.

IN PRICKLE-CELL CARCINOMA the cells of this layer become abnormally large, the connecting fibrils are lost, the basal-cell layer is penetrated by the downward growth and irregular proliferation continues in the corium.

SPONGIOSIS is a pathologic condition in the prickle-cell layer consisting of intercellular edema, enlargement of the intercellular spaces and an accumulation of fluid which, if under pressure may result in a vesicle. The roof of the vesicle usually consists of the overlying stratum corneum. Spongiosis is always associated with vascular dilation. It always is present in eczematous dermatitis.

PSEUDOEPIITHELIOMATOUS HYPERTROPHY is a benign hypertrophy of the epidermis characterized by microscopic features suggestive

of squamous cell carcinoma. The condition is found in deep fungus lesions such as blastomycosis, sporotrichosis and kerion, loderma and bromoderma late syphilis and the edges of old stasis ulcers. Diagnosis is made by the history microscopic studies of several biopsies and response to specific therapy

BALLOON DEGENERATION results from intracellular edema of the prickle cells with loss of prickles, vacuolization and separation of the cells from one another

The stratum granulosum, or granular layer is a narrow strip of flat rhombic cells containing keratohyalin a horny substance which is the precursor of keratin. In the presence of spongiosis, the resulting edema interferes with the normal production of granular cells and results in parakeratosis (scaling)

The stratum lucidum is a thin bright strip of nonnucleated cells which contain eleidin, an intermediate product between keratohyalin and keratin.

The stratum corneum is the outside layer of the skin and reflects changes from within as well as exogenous disturbances. It is subject to constant shedding and varies in thickness in different areas. This layer contains several rows of completely cornified hard, nonnucleated horn cells containing keratin and cell grease.

PARAKERATOSIS. If normal cornification is disturbed by a disordered prickle-cell layer partial cornification, or parakeratosis, results. This is clinically evident as a scale which is a loosened nucleated horn cell. Parakeratosis always occurs in psoriasis and other dermatoses characterized by scaling

HYPERKERATOSIS is an intensification of the process of cornification resulting in a thickening of the stratum corneum. The cells are closely adherent, nonnucleated, rich in keratin but poor in grease substance. Hyperkeratosis is usually associated with acanthosis and spongiosis. A callus is a familiar example.

DYSKERATOSIS is an abnormal disturbance of cornification resulting in vacuolization of the cells of the prickle-cell layer. The process may be benign as in Darier's disease or malignant as in Bowen's carcinoma which also is characterized by clumping of the cells and mitoses.

CUTIS

The cutis (dermis or corium) which is mesodermal in origin, is a continuation of the overlying epidermis. It extends upward,

dovetailing into the rete pegs of the epidermis as papillary projections, which are anchored by Herxheimer fibrils.

The corium consists of an upper papillary layer and a lower reticular layer which contain a latticework of elastic and collagenous fibers. The uppermost part, which lies between the rete pegs, consists of papillae which become widened flattened or elongated in pathologic conditions.

The corium also contains the important appendages of the skin, including the blood vessels, the lymph spaces the nerve endings, the sebaceous and the sweat glands, the hairs and their follicles and the arrectores pilorum (smooth muscle)

The corium is the site of the more serious cutaneous disturbances, including syphilis, the lymphoblastomas and the malignancies all of which are characterized by infiltrations. The presence of a perivascular or a cellular infiltrate may be nonspecific or diagnostic. The infiltrate may originate from the vascular or the lymphatic spaces or from the fixed connective tissue cells. It may consist of small round cells, or lymphocytes, polymorphonuclear leukocytes eosinophils, giant, mast or plasma cells. If the inflammatory infiltrate destroys the elastic and fibrous tissues, a scar or atrophy results.

SUBCUTANEOUS TISSUE

The subcutaneous tissue or hypoderm, consists of loose fatty tissue and elastic fibers. This layer protects the blood vessels and the nerves, provides for efficient heat regulation gives contour to the body and acts as a cushion against shocks. Gummata, tuberculous malignancy and foreign body reactions may occur in the subcutaneous tissue.

CUTANEOUS APPENDAGES

The appendages of the skin are described in their appropriate chapters.*

EMBRYOLOGY OF THE SKIN

The study of the fetal development of the skin and its appendages helps us to understand disturbances in the normal

*For a more detailed study of the subject, see Veredell, G. *Modern Trends in Dermatology* chap. The Anatomy of the Skin, New York, Hoeber 1943 Smith, P. E. and Copenhaver W. M. *Basley's Textbook of Histology* chap. 14 The Integument, Baltimore Williams & Wilkins, 1953 and Allen, A. C. *The Skin*, chap. 1 St. Louis, Mosby 1954

growth of the skin. It interprets anomalies and rudimentary structures which make their appearance at birth or soon after and explains the origin of certain benign and malignant tumors of the skin.

The epidermis, which is derived from the embryonic ectoderm appears first as a single row of cuboidal cells. A second layer of fine cells is visible soon afterward which forms the germinal



FIG. 2 (Top) Section from forehead of 4-month-old fetus. (Bottom) Section from scalp of 4-month-old fetus.

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PLATE 2



Syccosis vulgaris



Port-wine angioma



Tinea versicolor



Neurofibromatosis



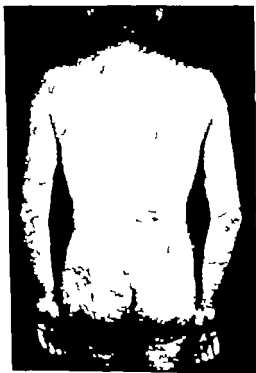
Pityriasis Rosea



Infantile Eczema



Impetigo Circinata

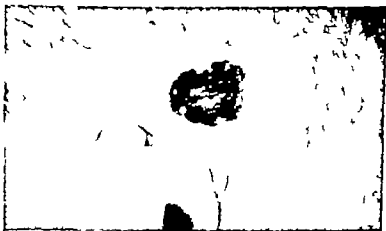


Ulcer Dermatitis

PLATE 5



Melanoma



Nevus Pigmentosus

(e.g., cutis elastica and epidermolysis bullosa) (4) ectodermal dystrophies (e.g., anidrotic ectodermal dysplasia), (5) metabolic disturbances (e.g., congenital porphyria) (6) disorders of pigmentation (e.g., albinism) (7) disorders of growth (e.g., neurofibromatosis) (8) hair anomalies (e.g., universal hypertrichosis, congenital alopecia) and (9) congenital fistulae and cysts.

NURSING ASPECTS

Unobstructed daylight is preferable for the complete examination of the skin. If not possible then blue fluorescent lighting is satisfactory. Unless the lesion or the eruption is localized, the patient should be asked to disrobe. The nurse provides clean sheets for the proper covering of the body until the physician is ready to examine the eruption. Female patients should be asked to remove brassieres, girdles, trusses, etc., in order to save time. Dressings should not be removed but the nurse should have benzene tincture of green soap light mineral oil or hydrogen peroxide at hand for this purpose. It is understood that the nurse should not volunteer any information regarding the cause, the treatment or the prognosis but a cheerful, sympathetic attitude is important.

The examination table should contain small towels, tongue depressors, cotton applicators, a flashlight, clean glass slides and cover slips, a drop bottle containing 20 per cent potassium hydroxide, a biopsy set, a small dermal curet, thumb forceps, red skin crayons, scratch pad cotton balls and a magnifying lens. A sterile syringe containing $\frac{1}{2}$ cc. of epinephrine is useful in case it is needed to counteract treatment emergencies.

Storage cabinets should be stocked with tubular gauze muslin strips, sterile gauze "hypo-allergenic" adhesive tape, biopsy bottles, history and treatment forms, gauze and elastic bandages, Band-aids of various sizes, eye pads stock lotions, paints, ointments and diagnostic sets for testing for contact dermatitis and allergies.

Before giving an injection the nurse should question the patient regarding local focal or general reactions from previous injections. She should not administer any injection, especially penicillin without orders from the physician.

The Erythema Group

THE HYPEREMIC ERYTHEMAS	SERUM SICKNESS
ERYTHEMA SIMPLEX	PAPULAR URTICARIA
ERYTHEMA PALMARE	PURPURA
INFLAMMATORY ERYTHEMAS	PURPURA SIMPLEX
INTERTRIGO	HENOCH SCHÖNLEIN PURPURA
NAPKIN ERYTHEMA	SYMPTOMATIC PURPURA
TOXIC ERYTHEMAS	PURPURA HEMORRHAGICA
ERYTHEMA SCARLATINIFORME	SECONDARY THROMBOCYTO-
ERYTHEMA MULTIFORME	PENIC PURPURA
ERYTHEMA NODOSUM	LUPUS ERYTHEMATOSUS
GRANULOMA ANNULARE	PITYRIASIS ROSEA
URTICARIA	NURSING ASPECTS

THE various diseases described under this heading comprise the largest group of dermatoses. A large number of them are closely related cutaneous reactions or responses to various internal or external stimuli rather than distinct entities. The clinical type of lesion depends on the nature of the cause, the anatomic site the circulatory state the degree of natural immunity the state of the defense mechanisms and the individual predisposition.

THE HYPEREMIC ERYTHEMAS

ERYTHEMA SIMPLEX

Erythema simplex is descriptive of one or more bizarre or Ir regular patches of erythema which appear at intervals anywhere on the skin. The cause is often difficult to determine but some of the cases have a psychosomatic basis. Vasomotor disturbances, urticaria and drug eruptions must be ruled out. Treatment depends on the cause and includes a light diet a mild astringent lotion and a saline laxative.

ERYTHEMA PALMARE

This condition is a persistent erythema of the palms sharply limited at the wrists and the sides of the hands.

Etiology The cause is systemic and the following conditions have been associated with it. pregnancy estrogenic deficiency hepatic cirrhosis, hyperthyroidism and protein deficiency

Differential Diagnosis. Contact dermatitis limited to the palms is unusual. Hyperhidrosis of the emotional type must be ruled out. Congenital keratoderma palmaris in children may begin as "red palms" with hyperkeratosis developing later in life.

Treatment depends on the cause.

INFLAMMATORY ERYTHEMAS

INTERTRIGO

Intertrigo (chafing) is a dermatitis caused by repeated friction between two opposing skin surfaces with retention of sweat in the skin folds. The affected parts are inflamed glazed or macerated by sweat. The thighs, the breasts, the groins, the toes and the axillary spaces usually are involved especially in obese individuals, and in the folds of fat babies. When it occurs between the toes or in the genitocrural region, a fungus infection should be suspected. Intertrigo is common in infants because of irritation from wet diapers.

Treatment consists of rest of the affected area separation of the parts with cotton pads and application of soothing powders. A good one to use consists of equal parts of corn starch, zinc oxide and boric acid. Extreme cleanliness is important in order to prevent secondary infection.

NAPKIN ERYTHEMA

Napkin erythema (Jacquet) or diaper rash is a dermatitis of the lower abdomen the genitals, the thighs, and the lower buttocks (diaper area) occurring in infants, characterized by an eruption of varying degree. The prominences are effected most, while the flexures usually are spared. The eruption may be erythematous, papular vesicular or even ulcerative. Pruritus often is intense and causes the infant to become irritable and restless.

Etiology These cases result from an irritation caused by the formation of ammonia in the diapers. This gas is formed by the decomposition of the urea in the urine by *B. ammoniogenes* or *Micrococcus ureae* (Bergey) in the stool. However when the ammoniacal odor is absent, and the flexures also are affected, the cause is more likely to be the presence of alkaline soaps or powders in the diapers as a result of improper washing and rinsing. Other

causes include the wearing of rubber diapers highly alkaline or acid stools excessive fat in the diet resulting in acidosis and the compensatory formation of ammonia, and uncleanness.

Differential Diagnosis

	<i>Napkin Erythema</i>	<i>Congenital Syphilis</i>	<i>Momibiasis</i>	<i>Seborrheic Dermatitis</i>
Affected parts	Prominences	Palms and sole of feet, mouth, etc.	Mouth may also be affected	Flaccid, especially axilla and neck
Others in family		Mother has positive Wassermann reaction		Mother or nurse may have seborrhea of the scalp
Laboratory data		Positive Wassermann reaction	Culture positive	
Type of lesions	Erythema, papules, vesicles or ulcers	Papules or bullae	Macules with thin scaly edges	Greasy scaly patches

Contact dermatitis from using detergents or Chlorox when washing diapers may resemble the condition. A change in laundry methods and the use of mild soaps with a simple shake lotion applied locally three times daily usually is effective.

Treatment

1. Change the diapers as soon as soiled.
2. Do not permit rubber panties to be worn.
3. The diapers should be washed with a mild soap and water followed by several rinsings to remove excess soap alkali.
4. Soak the diapers, crib sheets and undergarments in boric acid solution or Diaparene (complex salt of ammonium chloride) solution before drying them.
5. Do not use soap and water on the affected parts but clean them gently with olive oil.
6. The following lotion should be applied three times a day

Zinc oxide		
Talcum	61	100
Glycerin		5.0
Voform		1.0
Lime water q.s. ad		100.0

7. After the acute stage clears, use Diaparene or Hydrocortisone ointment three times daily.

- 8 Decrease carbohydrates in formula.
- 9 Use a superfatted soap in washing the diapers.
10. Methionine or thiamine may be used to inhibit the excess ammonia formation.

ERYTHEMA SOLARE

(Sunburn. See Acute Solar Dermatitis p 282)

FROSTBITE

(See Dermatitis Congelationis p 278)

TOXIC ERYTHEMAS

TOXIC ERYTHEMAS IN CHILDREN

Infants and young children may exhibit a nonfebrile generalized, macular eruption interspersed with annular lesions. The onset is always sudden with extensive symmetrical involvement within 24 hours. Prodromal symptoms may be poor appetite and listlessness. The inner surfaces of the arms and the forearms, the abdomen the posterior thighs the neck, the buttocks, and the dorsal surfaces of the feet are the usual sites.

Etiology Toxins absorbed from the intestinal tract or ear infections are the usual cause.

Treatment. Proteins in the diet should be restricted for a few days, an enema given to cleanse the lower bowel and a zinc oxide-starch lotion applied three times a day. Antihistamines in the form of Benedryl or Pyribenzamine elixir three times daily are useful. Within a few days the eruption begins to fade.

ERYTHEMA SCARLATINIFORME

Erythema scarlatiniforme is a recurrent toxic eruption resembling scarlet fever. At the outset, patients usually complain of mild constitutional symptoms. In most of the cases the eruption consists of large bright red erythematous areas on the limbs and the trunk which gradually spread until practically the entire body is involved. Desquamation with the formation of large flakes begins on the second day while the rash is still present. In extensive cases the hair and the nails may be shed.

Etiology The disease is a variant of erythema multiforme in the broad sense. Generally it is considered to be a cutaneous reaction to various gastro-intestinal and toxic conditions including follicular tonsillitis.

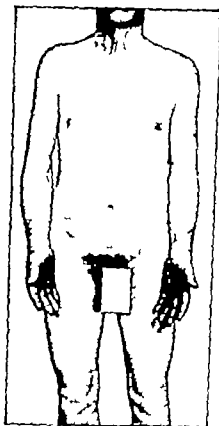


FIG. 3 Erythema scarlatiforme. Observe the asymmetrical distribution and diffuse involvement of the palms.

Differential Diagnosis

	<i>Scarlatiforme Erythema</i>	<i>Scarlet Fever</i>
History	Usually previous attack	None
Onset	Gradual no vomiting	Sudden with vomiting
Sore throat	Mild	Severe
Adenopathy	None	Present
Rash	Starts on limbs and trunk	Usually begins on neck and chest
Tongue	Denuded	Strawberry type
Duration	Several weeks	About 10 days
Desquamation	Starts on second day	Starts on seventh day
Albuminuria	None	Frequent
Leukocytosis	Absent	Present
Fever	Moderate	High
Schultz-Charlton test	Negative	Positive

Prognosis and Course. The disease which is rarely serious, usually persists from 5 to 14 days. Unfortunately relapses are common, but eventually immunity is established.

Treatment. Symptomatic relief is obtained by the use of calamine liniment N F. Bed rest and a trial of the broad spectrum antibiotics shorten the course of the disease.

ERYTHEMA MULTIFORME

Erythema multiforme is an acute toxic symmetric eruption or cutaneous reaction involving the face the extensor surfaces of the hands and the forearms, the palms the soles and the mucous membranes.

Clinical Description. The onset is sudden with or without symptoms of fever malaise joint pains, sore throat or diarrhea. Preceding the eruption there is often a history of a "fever blister"



FIG. 4 Erythema multiforme

in the mouth or on the lips. The presence at one time or another of bright red macular papular, gyrate, circinate discoid, bullous or urticarial lesions gives the eruption a polymorphous character. In some cases the eruption consists of large, edematous, purplish-red plaques. In others, a few bullous lesions may be present at the edge of a patch of erythema or the entire eruption may be bullous.

THE PATHOGNOMONIC LESION when present, is the fish-eye or target lesion consisting of one pinkish ring within another or an edematous macule with a central vesicle or bulla. Symptoms of marked burning and smarting are a feature of the disease. The primary lesion is an edematous macule.

THE MUCOUS MEMBRANES of the lips, the mouth or the tongue are involved in about half the cases, causing considerable pain and discomfort. When this occurs, the disease may be confused with early syphilis.

Clinical Types consist of idiopathic, symptomatic, erythema multiforme bullosum (page 149) and erythema perstans.

Etiology This disease is a cutaneous reaction of vascular hypersensitivity in predisposed individuals due to multiple causes. (1) bacterial proteins (acute rheumatic fever typhus, etc.) (2) drugs (sulfonamides, penicillin antipyrin) (3) viruses (vaccination herpetic) (4) focal infections, (5) toxins (after deep x ray therapy) and (6) undetermined. The idiopathic recurrent type is characterized by a seasonal incidence in the spring and the fall.

Pathology The main features, which are not diagnostic, are primarily in the corium, which contains dilated blood vessels with edematous walls dilated lymphatics and an infiltrate composed of leukocytes and eosinophils in the early cases, lymphocytes in the late cases. The changes in the epidermis are secondary with intracellular and extracellular edema and vesicles the size of which depend on the acuteness of the disease.

Prognosis. Erythema multiforme is a self limited disease. It may extend over a period ranging from 10 to 14 days although recurrences may come at intervals.

Treatment

1 For the severe cases consultation with a dermatologist is advisable.

2 Search for and eliminate all foci of infection in the interval between attacks

3 ACTH AND CORTISONE. In severe cases, the patient should be hospitalized and 10 mg. ACTH given by intravenous drip daily for several days until the lesions dry up. Then a maintenance dose of 25 mg. cortisone daily for a week or longer should be prescribed.

Cortisone 300 mg. I.M. the first day 200 mg. the second day and then a daily injection of 100 mg. for a week or longer also may give satisfactory results.

4 LOCAL TREATMENT. The erythematous types respond well to calamine lotion. When the lips are involved compound tincture of benzoin is useful. Astringent mouthwashes (1:5 Burow's solution) or the local application of milk of bismuth or magnesia are recommended in the oral cases.

5 INTERNAL TREATMENT. The standard treatment in the idiopathic cases is the administration of salicylates. In febrile cases a course of the broad spectrum antibiotics for several days is worth a trial. Avoid sulfonamides.

6 COLLATERAL TREATMENT. Autobemotherapy or 5 per cent intravenous dextrose injections are useful procedures in some cases. The management of the extensive cases includes complete bed rest, a salt free diet and the use of colloidal baths, which tend to dry up the lesions and afford the patient much relief.

ERYTHEMA PERSISTENS is a general term used to describe the persistent types which involute very slowly.

Erythema centrifugum annulare, an eruption of large, bright red edematous macular lesions, characterized by centrifugal spreading is included in this group. These usually occur on the trunk and form an annular or gyrate pattern with raised and thickened edges. After the lesions disappear they may leave pigmentation behind.

Erythema figuratum persistens, *erythema simplex gyratum* and *erythema chronica migrans* are all variations of the persistent type of the disease.

Etiology. Most cases occur in apparently healthy persons, but in rare cases the dermatosis may be associated with visceral malignancy, chronic arthritis and intestinal toxemia. Some authors regard *erythema annulare centrifugum* as a dermatophytid. Lesions similar to *erythema migrans* may rarely occur at the site of insect bites.

Differential Diagnosis. Persistent erythema must be differen-

tiated from delayed port wine nevus, chronic actinic dermatitis and fixed drug eruptions.

Treatment is that of the cause.

ERYTHEMA NODOSUM

Erythema nodosum is an acute inflammatory symptom complex due to various sensitizing or antigenic agents in a sensitized individual and is characterized by the appearance of painful subcutaneous nodules with or without systemic disturbances. The disease consists of a symmetrical eruption of subcutaneous, hard red oval nodules on the shins, the extensor surfaces of the arms and the buttocks. The eruption begins to involute soon after reaching its peak. After two or three days the lesions become softer with gradual color changes ranging from bright red, to violet, to yellow (bruise cycle). As a rule, they continue to come out in crops for periods of several weeks. The lesions of erythema nodosum never ulcerate or break down characteristics which distinguish them from the lesions of erythema induratum. Occasionally the eruption is associated with erythema multiforme. The nonspecific types usually are associated with a hilar adenopathy which disappears with the clearing of the skin lesions.

Etiology Erythema nodosum is a disease of early adult life most of the cases occurring in the 10- to 30-year age group. There is a seasonal incidence with the peak extending from November to March. Since the disease is a symptom complex, the

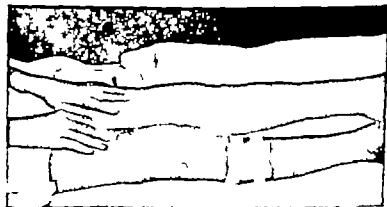


FIG 5 Erythema nodosum. Note the tense shiny appearance of the lesions. (Dr A W Nelson's case)

cause varies in individual cases, even as to geographic factors. The infectious types may be caused by various organisms. *Streptococcus haemolyticus* often is found in cultures from the nose and the throat in adult cases. European investigators regard the tubercle bacillus as an important factor in juvenile cases, but American dermatologists consider the presence of this organism as that of a secondary invader due to lowered resistance.

Erythema nodosum may occur in connection with the following diseases: fungus infections, malaria, gonorrhea, brucellosis, early syphilis (with or without syphilitic phlebitis), granuloma inguinale, lymphogranuloma venereum, leprosy, sarcoidosis, Behcet's syndrome, coccidioidal granuloma, Rocky Mountain spotted fever and drug eruptions (sulfathiazole, iodides and bromides). In some cases there is no apparent cause for the recurrent attacks.*

Pathology. The corium is invaded by a dense perivascular infiltration containing polymorphonuclear leukocytes. Pressure from the dense infiltrate may result in the formation of necrotic areas. Thrombosis is often present as a result of bacterial emboli. Many of the blood vessels are dilated and show inflammatory changes in their walls.

Prognosis. Erythema nodosum is a self limited disease. Recurrences are common. Individual attacks may last longer than two or three weeks.

Differential Diagnosis. Erythema induratum is chronic, not painful, attacks the backs of the legs and always ulcerates. Nodular vasculitis consists of tender nodules that extend along the course of the superficial vessels of the anterior surface of the legs. The patients are in good health—the lesions do not go through the bruise cycle—new lesions persist for several months—and the perivascular infiltrate consists mainly of lymphocytes.

Treatment. If the eruption is extensive, the patient should be put to bed and a light diet prescribed. Antibiotics, acetylsalicylic acid, sodium salicylate or sulfadiazine should be prescribed in febrile cases. Local treatment consists of cold boric packs or wet dressings of 0.5 per cent aluminum acetate. If the eruption does not respond to these measures, a course of cortisone for 5 days may be effective if there are no contraindications.

In view of the fact that the disease represents a cutaneous reaction to a focus of infection, the patient should be examined

*For an excellent review of the subject, see Beerman, H. Erythema nodosum. Survey of the recent literature, Am. J. Med. Sc. 223:433, 1952.

genitalia. If edema of the glottis occurs, strangulation is apt to result. A similar type of reaction may occur in the lungs and the intestinal tract.

Etiology The disorder is due to various causes which activate histaminelike or acetylcholine antigens. These act directly or indirectly on the walls of the capillaries and produce changes in the permeability and the size of the vessels as well as the rate of flow. The following are the most important.

1 **FOOD ALLERGY** Practically all the acute cases are due to a sensitization of the cells in the upper cutis where an antigen-antibody reaction occurs. Fish seafoods, pork veal, strawberries, nuts and mushrooms are common allergens. Excessive beer drinking may be a factor in some of the chronic cases.

2 **DRUG ALLERGY** The drugs which usually cause chronic urticaria include penicillin quinine the sulfonamides, salicylates and barbiturates.

3 **PROMOTIVES** Acute attacks characterized by gastro-intestinal symptoms usually are due to spoiled foods, with the production of cholinelike amino acids.

4 **TOXINS IN THE BLOOD STREAM** The chronic cases are produced by foci of infection absorption of abnormal protein fractions (gastro-intestinal disease) menstruation pregnancy biliary disease, nephritis, fungus diseases, glycosuria, syphilis and other organic diseases.

5 **SERUM AND VACCINE REACTIONS.** Antibiotics, liver injections, estrogens and various vaccines and sera may produce acute or chronic reactions in susceptible patients.

6. **THE EMOTIONS** may act as foci of irritation and produce chronic urticaria by direct (shock) or indirect (effect on digestion) action. These disturbances include high tension chronic financial or domestic worries, sex maladjustment, fatigue, excessive mental exertion or vagotonic states associated with the menopause with resulting vasomotor imbalance.

7 **ACIDITY HYDRIA.** Since many chronic conditions are associated with a disturbance of the gastric secretions, a low hydrochloric acid content may be found in as high as 65 per cent of the cases. This type of case responds to therapeutic doses of dilute hydrochloric acid.

8 **PHYSICAL CAUSES** Exposures to cold temperatures, cold baths, intense heat, sunlight or friction (girdles etc.) occasionally may produce urticaria. This type of eruption, which usually

appears on the exposed surfaces, results from the formation of acetylcholine.

9. **INHALANTS** such as perfumes orris root feathers, strong odors, etc.

10. **TRANSEPIDERMAL SENSITIZATION** to an absorbed substance in a contact dermatitis.

11. **PARASITIC DISEASE**, especially amebiasis and roundworms in children.

12. **INSECT BITES**.

13. **ENDOCRINE DYSFUNCTION** including hypothyroid states, hyperthyroidism menstruation, pregnancy and Fröhlich syndrome.

14. **AUTOGENOUS SENSITIZATION** to an excess of normal substances (acetylcholine, extravasated blood etc.) may cause chronic types.

Diagnosis. The characteristic fleeting character of the eruption, the marked itching and the presence of wheals are pathognomonic. When urticaria is due to food idiosyncrasy the eruption disappears after several days when the suspected food is eliminated. If the eruption still persists a careful history should be taken to eliminate drugs, emotional disturbances and other causes mentioned under etiology. When allergy to foods is suspected elimination diets should be prescribed in an effort to determine the particular food which may be causing the urticaria. Skin tests are of no value during attacks because of the reactive capacity of the skin to trauma resulting in false-positive tests. Urticaria which occurs at night may be caused by an allergy to substances in the bedding. Urticaria resulting from physical allergy usually consists of large wheals on the exposed surfaces and is often asymmetrical.

In the chronic types the patient should carry a notebook to record the circumstances of each attack in an effort to determine the cause (parallel bookkeeping). Focal infection, visceral disease blood dyscrasias, internal tumors, pyorrhea, intestinal parasites and psychosomatic influences, should be investigated in those cases which persist in spite of symptomatic therapy.

Differential diagnosis is between insect bites and scabies. Insect bites are characterized by grouping, presence of fixed papular lesions, central punctum presence of a few hemorrhagic lesions and nocturnal itching. Scabies involves the characteristic sites there may be other cases in the family and tiny vesicles are present.

Prognosis. Most cases of the acute type caused by food allergy clear up within a few days. Those resulting from penicillin sensitivity may last for several weeks in spite of rational therapy. Chronic urticaria may respond temporarily to Benedryl or Pyribenzamine, but until a specific cause is found and eliminated the eruption persists, its severity changing from day to day until eventually it disappears spontaneously.

Treatment. **ACUTE CASES** 1 *Rest* If the eruption is at all extensive bed rest is important to permit the natural immunologic defenses to develop.

2 *Saline purgatives* should be administered at once to remove intestinal putrefaction even though the patient is not constipated.

3 *Vasoconstrictors* For immediate control, adrenalin is administered by injection if there are no contraindications (hypertension, myocarditis and rapid pulse). Small doses act much better than large doses. Adrenalin in oil which maintains a prolonged vasospastic effect from 9 to 16 hours, is preferable in some cases. When a milder action is desired ephedrine sulfate combined with an antihistaminic may be prescribed.

4 *Sedation* If the patient is high strung, restless and complains of inability to sleep as a result of the severe itching, relief can be obtained in some cases from a substantial dose of one of the antihistaminics, phenobarbital or bromide sedation. Under no circumstances should codeine or morphine be used.

5 *Antihistaminics* These drugs control the disease in about 75 per cent of the acute cases. Benedryl is the most effective but a trial of 3 or 4 days with similar compounds is warranted if results are not obtained. Smaller doses are given during the day with maximum dosage at bedtime. There is no advantage in giving these drugs parenterally. It must be emphasized that these drugs are only suppressive and not curative so that each case requires an evaluation of possible etiologic factors. The patient should be warned of the possible side-effects and one of the amphetamine drugs should be prescribed along with the drug to combat daytime sedation. Occasionally paradoxical reactions result with an increase in the number of lesions.

6 *Local Therapy* A great deal of comfort is derived from the colloidal bath (see p. 599) which should be taken in the morning and at bedtime. Calamine and zinc oxide lotions com-

taining menthol phenol or liquor carbonis detergens are indispensable for temporary relief

7 *Elimination Diet* In acute urticaria where food allergy is suspected, avoid the following fish of all kinds, chocolate and cocoa, fresh fruits and juices, beer oatmeal mushrooms nuts pork ham, bacon lard Jell-o and cola drinks.

CHRONIC CASES Until the cause is found and eliminated the following drugs may be of service 1 *Intestinal antiseptics* including sodium ricinoleate bile salts, sulfaguanidine salol *Bacillus acidophilus* and brewer's yeast are of value. *B. acidophilus* liquid is prescribed in doses of 2 or 3 tablespoonfuls mixed with 2 tablespoonfuls of lactose in milk.

2 *Gynergen* for its vasoconstrictor effect and *Bellergal* for its antispasmodic action may be effective in menopausal women, but side-effects may interfere with their continued use.

3 *Hydrochloric Acid* Since some urticarial patients may have a low hydrochloric acid gastric analysis should be a routine procedure in the chronic cases. If the acid content is low Acidulin ($\frac{1}{2}$ hour after meals) should be prescribed

4 *Anticholinergic drugs* Those cases due to psychosomatic and physical causes may respond to Antrenyl Bromide (Ciba) Pro-bathine (Searle) Pamine Bromide (Upjohn) or Co-Elorine (Lilly)

5 *Foreign protein therapy* is effective sometimes in the chronic cases, although its action is erratic and indeterminate. Autohemotherapy and autogenous vaccines made from stool cultures or milk injections every 5 days can be used in selected cases.

6 *Calcium* Intravenous injections of Neo-Calglucon (Sandoz) may be of temporary benefit.

7 *ACTH* and *hydrocortisone* may give temporary relief but their use should be restricted to unusually severe cases.

8 *Psychotherapy* is important in all cases with a psychosomatic background.

9 If the eruption is aggravated by any of the above measures, the best advice is to discontinue all therapy. Sometimes the results are surprising.

SERUM SICKNESS

Serum sickness is an anaphylactic reaction following the injection of penicillin or animal or human foreign serum. Usually

It is preceded by an incubation period of from 7 to 12 days. The eruption often is urticarial or erythematous, although angioneurotic edema of the lips, the eyelids, or the extremities is not uncommon.

The symptoms consist of fever ranging from 101° to 102° F., generalized adenopathy joint pains, malaise and a transient albuminuria. Extensive cases persist in mild form for several weeks.

Treatment consists of antipruritic lotions, injections of Benedryl (2 to 5 cc.) t.i.d. colloid baths and rest in bed. ACTH and cortisone are effective in the early cases and should be followed up with antihistaminics to avoid relapses.

PAPULAR URTICARIA

Papular urticaria (lichen urticatus) is a recurrent, pruritic, papular eruption occurring in children during the summer months.

Clinical Appearance. The eruption consists of generalized evanescent pink wheals in the center of which there is a pinhead papule. There is no characteristic distribution or grouping, which at first tends to confuse the examiner. The distinctive wheal surrounding the lesion appears during the night but disappears quickly leaving the papule, the top of which usually is excoriated from scratching. In acute cases the eruption tends to be papulovesicular. Severe cases often are complicated by excoriations from scratching and secondary infection leading to impetigo furuncles, excoriations, crusts and pigmentation. Enlargement of the lymph nodes is a frequent finding in cases of long standing. The itching which is present both day and night, is relieved temporarily by scratching. This reflex removes the surface of the lesion, resulting in a lessening of intrapapular tension. Dermatographism is found in some cases, but true urticarial lesions are seldom present.

Etiology. Papular urticaria is limited to children. The age incidence is from 2 to 8 years. Chronic types may be due to a reaction of hypersensitivity to unknown antigens. The cases occurring in the summer months have been shown to be caused by insect bites (Goldman).

Prognosis. Those cases beginning in the second half of the first year usually persist until the third year. In rare cases the disease may occur at irregular intervals up to puberty.

Differential Diagnosis

	<i>Psoriasis Urticaria</i>	<i>Scabies</i>	<i>Chickenpox</i>	<i>Insect Bites</i>
Distribution	Face, buttocks, extremities	Fingers, wrists, trunk, buttocks	Face scalp trunk, mouth	Exposed parts. Central punctum
Mouth lesions	None	None	Yes	None
Season	Summer	Winter	Fall and winter	Summer
Eosinophilia	Slight	Yes	No	No
Symptoms	Irritability itching day and night	Itching, worse at night		Stinging

Prurigo mitis, which is rarely seen in this country occurs more frequently on the legs itching is more intense, undernourishment is present, adenopathy is common, residual pigmentation is frequent, and chronicity is the rule.

Treatment. The object of the treatment is to reduce the itching and to prevent secondary infection. All sources of psychic, toxic and focal irritation should be removed. The local treatment consists of antipruritic lotions and colloidal baths (see p. 599) to relieve the itching. Rapid response usually follows (1) injections of parathyroid hormone, which probably acts by increasing the calcium in the blood stream, thus relieving the pruritus, (2) intramuscular injection of mother's blood (5 to 10 cc.) (3) shake lotions containing $\frac{1}{2}$ 1 per cent phenol and (4) the antihistaminic drugs (see p. 606) to control the itching. The bedroom should be well screened to avoid the possibility of insect bites. DDT should be used on the premises for prophylaxis.

PURPURA

In this large group of important syndromes there is extravasation of blood into and beneath the skin and in the mucous membranes.

The primary lesion is a nontraumatic purpuric spot that may change its size, color or shape depending on various conditions. It is characterized by (1) acute onset (2) variability in the size of the lesions, depending on the cause and the site of the eruption, (3) variegated color changes depending on the age of the lesion and (4) persistence of the lesions, which do not disappear on pressure. The smallest purpuric lesions, called petechiae, are

round pin point macules. Ecchymoses are large irregular lesions, ranging from 5 to 15 cm in size. Vibrioes are linear hemorrhages. When the hemorrhage occurs in the subcutaneous tissue a raised tumor is produced. It is called a hematoma.

Clinical Types. The study of a case does not rest with the diagnosis of purpura, but the etiology must be investigated by laboratory procedures, a thorough physical examination and a complete history of the case. The nonthrombocytopenic cases are characterized by a normal platelet count. The thrombocytopenic cases of purpura by a decrease in the blood platelets.

Diagnosis. A bilateral eruption of purplish macules, which do not fade on pressure should suggest purpura. In atypical cases and in those refractory to therapy a biopsy with special staining for iron pigment or a sternal puncture to rule out the lymphoblastomas is necessary.

Pathology. is fairly uniform in all types with normal epidermis dilated capillaries and deposits of hemosiderin about the blood vessels in the corium.

Laboratory tests. The following tests should be made on all cases of purpura clotting time bleeding time clot retraction prothrombin time capillary resistance and platelet count.

PURPURA SIMPLEX

Purpura simplex is a common form of primary nonthrombocytopenic purpura due to various causes. The presence of a normal platelet count distinguishes it from the more serious forms of the disease. The mild forms usually affect children and rarely last longer than 2 to 4 weeks. The disorder sets in with or without malaise anorexia and joint pains, although occasionally slight anemia and fever may be associated with the condition. In most of the cases the eruption occurs on the feet, the inner thighs the trunk the arms and the forearms.

Etiology. The cause is often obscure but acute gastro-intestinal upsets and psychosomatic factors may provoke the disturbance.

Prognosis. Most cases clear up within 4 weeks. Recurrences are not uncommon.

Treatment consists of bed rest rutin ascorbic acid and a nourishing diet including citrus fruits raw cabbage and tomatoes, which are rich in vitamin C. A search should be made for the

cause including drugs, focal infection malnutrition intestinal parasites and auto-intoxication.

HENOCH SCHÖNLEIN SYNDROME

The Henoch-Schönlein syndrome is a type of anaphylactoid primary nonthrombocytopenic purpura usually occurring in children and characterized by a polymorphous eruption. Any of the lesions of the erythema group may be present, including purpura, urticaria, erythema and bullae. In most of the cases such internal disturbances as abdominal colic, fever, bloody



Fig. 7. Purpura simplex.

stools, diarrhea and vomiting are present. Arthritis and nephritis with hematuria may result from hemorrhagic foci. There are apt to be ocular hemorrhages or bleeding from the nose, the throat, the mouth or the stomach in the extensive cases.

Etiology is not proved definitely. Some cases appear to be caused by a streptococcal focus; others appear to be an allergic type of reaction or endothelial sensitization to various foods. These include wheat, eggs, milk, chocolate, nuts, beef, fish or onions. Elimination diets should be prescribed in an attempt to determine the offending foods.

Diagnosis is based on the following points: (1) evidence of abnormal capillary bleeding of the skin and the mucous membranes, (2) normal platelet count, (3) absence of any known cause for the bleeding, (4) laboratory tests and (5) acute arthritic or abdominal symptoms (colic, vomiting and melena).

Course. Recovery is the rule, although some cases persist indefinitely with remissions and exacerbations.

Treatment consists of bed rest, elimination of the suspected foods, cortisone or ACTH until the bleeding is arrested, blood transfusions in the severe cases, vitamin C medication, anti-histaminics and a nourishing diet.

SYMPTOMATIC PURPURA

Symptomatic purpura (secondary nonthrombocytopenic) comprises a large group of conditions characterized by a purpuric eruption without platelet changes. A history, a physical examination and tests to determine the presence or the absence of diseases of the blood-forming organs are necessary to arrive at a proper interpretation.

Etiology. The causes of symptomatic purpura include the following:

1 **INFECTIOUS DISEASES** (usually of serious significance). Bacterial endocarditis, scarlet fever, smallpox, measles, cerebrospinal meningitis, typhus fever, acute lymphatic leukemia and lupus erythematosus disseminatus.

2 **TOXIC PURPURA.** Drugs, including the anaphenamines, salicylates, Dilantin, antihistaminics, antiheparin compounds, penicillin, quinine, iodides, sera and the sulfonamides.

3 **ALLERGIC PURPURA.** Chocolate, nuts, fish, berries, etc.

4 **ENDOCRINE.** Menstruation, pregnancy.

5 MECHANICAL. Venous stasis, septic emboli malignant hypertension mild trauma from rubbing whooping cough, epilepsy

6 HEPATIC. Diseases of the liver (acute and chronic)

7 CACHECTIC. Senility carcinoma, nephritis, tuberculosis.

8 BLOOD DYSCRASIAS. Leukemia, scurvy pernicious anemia and aplastic anemia.

9 ACTINIC. Purpura solaris may result from exposure to intense sunshine as a result of degenerative changes in the capillary walls. Usually the backs of the hands and the legs are affected.

10 INSECT BITES may produce a localized purpura in susceptible patients.

Treatment. The cause should be discovered if possible and combined rutin-scorbic acid prescribed in adequate doses. Bed rest and intravenous glucose are important collateral measures.

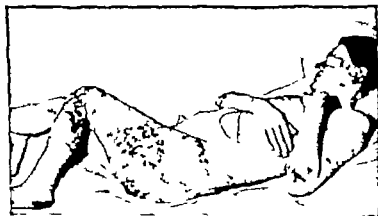


FIG. 8. Purpura hemorrhagica. Note the outline of the enlarged spleen.
(From Dr. J. P. Long)

PURPURA HEMORRHAGICA

Purpura hemorrhagica is a serious type of bleeding in the skin and the mucous membranes. A marked reduction in the platelet count, secondary anemia and a disturbance of the blood forming organs are also present. The disease not only is characterized by a purpuric eruption which consists of petechiae, purpuric macules and ecchymoses, but hemorrhages frequently occur in the mucous membranes and the internal organs. Bleeding from the nose or the mouth may reach alarming proportions.

Etiology is unknown. Some investigators believe an antibody that has a platelet-destroying effect is produced in response to sensitization by an unknown factor. It is difficult to determine whether the disease is due to increased destruction or decreased output of blood platelets.*

Hematology The blood platelets are reduced (normal—120,000) the bleeding and the coagulation time are increased and capillary fragility is pronounced. Clot retraction is usually absent. There is lessening of capillary resistance as demonstrated by the tourniquet test on the arm and the thigh.

Differential Diagnosis. Pernicious anemia, leukemia and aplastic anemia must be eliminated by repeated hematologic and bone-marrow studies.

Prognosis is unpredictable. Splenectomy results in cures in about 80 per cent of the cases, while a few clear up spontaneously.

Treatment. Palliative measures include (1) horse serum (2) intramuscular blood injections (3) complete bed rest (4) vitamin K if prothrombin is low (5) frequent blood transfusions to control the secondary anemia, (6) moccasin venom, (7) rutin.

If the bleeding continues for one week in spite of these measures, splenectomy must be performed provided that the patient is a good risk. Hydrocortisone or ACTH alone or in conjunction with splenectomy has been effective as a curative measure in many cases.

SECONDARY THROMBOCYTOPENIC PURPURA

This is a disturbance of the bone marrow from various causes which interfere directly with the production of blood platelets. The eruption and the blood picture are similar to those of purpura hemorrhagica.

Etiology includes (1) tumors of the bones, involving the bone marrow (2) blood diseases (3) drugs (benzene, gold, Sedormid, propylthiouracil, sulfonamides, arsenicals) (4) severe infections (septicemias) (5) radioactive substances (prolonged exposure to radium, radioactive phosphorus or x-rays) and (6) nitrogen mustards.

*See Luzzati, E. L. Current status of pathogenesis and treatment of thrombocytopenic purpura. *Quart. Rev. Med.* 9:61-72, 1952.

Diagnosis. A complete history a physical examination blood and bone-marrow studies and roentgenograms of the bones are necessary in order to obtain an accurate diagnosis.

Treatment is ineffective unless the underlying cause is determined. Repeated blood transfusions, ACTH and cortisone are of temporary value.

LUPUS ERYTHEMATOSUS

Lupus erythematosus, or butterfly lupus, is a capricious systemic disease with skin manifestations. The latter consists of well-defined inflammatory patches characterized by redness and scaling. This often is followed by visible atrophy and a blotchy pigmentation.

Classification is based on the cutaneous features and the degree of systemic involvement (1) the chronic, localized or fixed type (2) the subacute and (3) the acute generalized or disseminated type. Whether they are distinct diseases, mutations or clinical varieties of the same condition cannot be answered with certainty in the present state of our knowledge. At present, however opinion regards chronic lupus erythematosus as a local manifestation of a generalized vascular disorder *

The Localized Types. CHRONIC DISCOID (FIXED TYPE)
This is the common variety which favors the flush areas of the face where the blood vessels are more resistant to vasoconstriction. The disease begins as a small scaly macule which fades on pressure, not unlike that of seborrheic dermatitis. After a variable length of time it enlarges to form a patch with a red elevated hyperemic and thickened border and a slightly depressed center. The surface is either smooth or covered with dry firmly adherent scales. As the older patches increase in size they often assume a reddish-violet color a variable degree of atrophy pigmentation and telangiectasia.

The typical lesions are characterized by well-defined borders and slow extension. Ulceration never occurs. Although central atrophy usually develops in the older lesions this is not essential in making a diagnosis. If the disease involves hairy areas, permanent alopecia results from follicular atrophy.

*For discussion of the modern approach to the subject see Michelson, H. E. Review and appraisal of present knowledge concerning lupus erythematosus, *AMA Arch Dermat & Syph* 69:694, 1954.

Localization The discoid variety is usually limited to the face. In most cases it first appears on the upper half of the cheek and gradually spreads across the bridge of the nose until both cheeks are involved giving rise to the characteristic butterfly appearance. Other areas affected are the ear the scalp the upper third of the chest, the tip of the nose the shoulders or



FIG. 9. Lupus erythematosus (common fixed, or localized, type). This is the chronic form. Note the atrophy and the butterfly distribution.

the vermillion border of the lips. In rare cases, the disease may be confined to the scalp.

The mucous membranes are involved in about 10 per cent of the cases but rarely without associated skin lesions. It is not unusual to find involvement of the vermillion borders of the lips. When they are attacked, they are at first somewhat swollen and violaceous later becoming atrophic, scaly and silvery. They sometimes appear to have been painted with collodion. In cases of upward extension from the cheeks, the lower eyelids or the conjunctival surfaces may be affected.

Mucous membrane involvement of the cheeks the palate or the larynx may resemble leukoplakia. There is little discomfort the lesions may be present for months or years before they are recognized. The buccal lesions favor the area where the upper and the lower molars meet. In the beginning the lesions are punctate bright red spots which later join to form irregular superficial erosions surrounded by a zone of hyperemia. After a variable length of time the center of the lesion becomes atrophic, but the border retains its hyperemic zone. Old patches are characterized by irregular white atrophic areas.

THE SUPERFICIAL TYPE consists of dusky red patches with very little scaling slight infiltration and slight or no clinical evidence of atrophy.

THE EDEMATOUS TYPE consists of pink edematous plaques that later assume the characteristic clinical features.

THE TELANGIECTATIC TYPE consists of circumscribed areas of redness which are due to a collection of dilated capillaries. The condition is symmetrical and involves both cheeks. Some cases resemble localized patches of poikiloderma.

LUPUS FERNIO is a form of lupus erythematosus which resembles the raised violaceous plaques of frostbite. The superficial Hutchinson type follows chilblain. The rare Bemler type (true tuberculosis) is associated with papulonecrotic tuberculids. The lesions in this type affect the ears, the fingers and the toes.

LUPUS ERYTHEMATOSUS PROFUNDA is a deep variety and probably related to sarcoid.

Etiology The cause of this unusual cutaneous-vascular disease is unknown. Females are affected more often than males. Most of the cases begin in the 20- to 30-year age group. The following theories are concerned in the etiology of the condition: (1) photosensitization (2) bacterial vascular allergy (streptococcus) (3) vascular response to cold, and (4) circulatory defects. Michelson* does not consider tuberculosis an etiologic factor or that light sensitivity plays an important role.

The best opinion at present regards lupus erythematosus, not as a clinical entity but as a vascular disease with skin manifestations. If it is a blood-borne disease, it probably is a toxemia rather than a bacteremia. The type of cutaneous response brought forth by these unknown etiologic agents depends to a

*Michelson, H. E. The problems of lupus erythematosus, Minnesota Med. 36 1043, 1943.

great extent on the constitutional background and the state of the vascular system.

Pathology A study of the histologic sections reveals a typical pathologic picture. In the discoid variety a characteristic feature is the keratotic plugging of the follicular openings. In the upper corium there is a perivascular infiltration of lymphocytes and dilatation of the capillaries and lymph spaces. In old cases, there are areas of beginning atrophy and loss of elastic tissue. No tubercles are present.

Prognosis and Course The fixed or discoid variety is characterized by a very slow course during which the lesions continue to spread and to involute spontaneously over a period of several years, resulting in various degrees of scarring pigmentation superficial scaling and alopecia of the hairy parts. With the advent of Aralen about 95 per cent of the chronic cases are benefitted. The atrophy of course is permanent.

A change in the inflammatory character of the lesion or a sudden or gradual *extension* of the eruption announces the onset of the acute or the subacute disseminated type. This usually is accompanied by fever chills, arthritic pains and symptoms of a toxic nature.

Diagnosis. The following characteristics are useful in making a diagnosis of chronic lupus erythematosus (1) Localization on face (nose cheeks, ears or scalp) (2) the typical lesion an erythematous scaly macule (3) removal of scale reveals patulous follicular openings (4) superficial atrophy present (5) slow evolution (6) absence of L E cells in the blood

Differential Diagnosis. Seborrheic dermatitis, when limited to the nose or cheek may cause confusion, but in this disease the patch is not sharply limited the scales are not adherent but are easily removed and greasy usually the scalp also is affected, infiltration and atrophy are lacking

Rosacea is more diffuse papulopustules are present as well as some telangiectasia

Lupus erythematosus of the scalp must be differentiated from folliculitis decalvans by the presence of typical lesions on some portion of the face and the absence of folliculitis at the borders of the atrophic patch

Phenolphthalein eruptions on the face in Negroes may cause confusion.

Treatment **CHRONIC TYPE** Before instituting therapy, a complete examination should be made including a chest roentgenogram and a blood count erythrocyte sedimentation rate and "L.E. cell" blood studies to rule out evidence of systemic involvement. The patient should be warned against exposure to the sun because of the danger of dissemination of the disease.

Local Therapy Irritating applications do more harm than good. Superficial lesions that do not respond to Atabrine or Aralen can be destroyed with applications of solid carbon dioxide slush but this does not prevent the development of new lesions or extension.

Chloroquine (Aralen) is the drug of choice. A 250-mg tablet is given twice a day for the first week and then reduced to once a day for several weeks or until the lesions fade. A maintenance dose of 1 tablet every other day may be necessary during the summer months. The only side-effect of importance is inability to accommodate. Carboquin and Primaquine are less toxic but the response is slower.

Quinacrine (Atabrine) may be prescribed if chloroquine is ineffective after several weeks. The objection to the drug is chiefly the yellow discoloration of the skin which occurs after a week or two, and the possibility of generalized lichen-planus eruptions.

Mucous membrane lesions may slowly respond to Atabrine or Aralen therapy after several months.

Systemic Types. The subacute and the acute types of lupus erythematosus are described separately because the clinical picture the laboratory findings, the prognosis and the treatment are different from those of the chronic type.

SUBACUTE LUPUS ERYTHEMATOSUS

Clinical Picture. The eruption may follow the chronic type after excessive exposure to the sun or it may appear *de novo*. The lesions are ill-defined more or less persistent, erythematous plaques with minimal scaling and more or less itching or burning.

The lesions usually affect the nose the ears, the neck, the upper back or the arms. A generalized adenopathy may be present. The main point to remember is that the patient looks and feels well in spite of the fact that we may be dealing with a mild form of the systemic type.

Laboratory data may be variable. In most cases all examinations are normal except for a slight anemia. If the sedimentation

rate is increased, if the anemia is progressive if the Kahn is falsely positive if the afternoon temperature is elevated, and a few lupus erythematosus cells are found in the peripheral blood, the patient is entering the acute stage.

Differential Diagnosis. Toxic erythemas, acute dermatomyositis and seborrheic dermatitis must be ruled out.

Pathology There is no specific picture. The superficial capillaries and the lymphatics are dilated with moderate edema in the upper cutis and liquefaction degeneration of the basal-cell layer



FIG 10 Acute lupus erythematosus with involution of the lips. The white cell count ranged from 2,500 to 3,800. The extensor surfaces of the elbows and the knees were the site of erythematous plaques.

Prognosis. The majority of the cases clear up spontaneously, but about 10 per cent develop into the acute type.

Treatment. The main object is to prevent the onset of the acute type. Locally calamine-type lotions are soothing. Chloroquine (Aralen) ascorbic acid and rutin are often effective systemically. To avoid excessive sun exposure a protective cream should be used (15 per cent para-aminobenzoic acid or 5 per cent Cycloform).

Rest is important, although hospitalization may not be necessary if the patient can be observed at frequent intervals.

ACUTE SYSTEMIC LUPUS ERYTHEMATOSUS

This syndrome occurs in two forms—one with skin manifestations, and one without skin lesions. There are three types of the disease: (1) the type that appears on the face as a result of irritation of the chronic type, (2) the recurrent type, consisting of episodes of arthritis, fever and chills, (3) the acute fulminating type with high fever and chills and symptoms of septicemia from the beginning.

The eruption usually starts on the face and spreads rapidly to the chest, the arms, the joints of the fingers, the knees and the ankles. The characteristic lesion is a bright red or pink, erythematous lesion. Later these may become purpuric or bullous.



FIG. 11 (Left) Lupus erythematosus. Note atrophy, alopecia and inflammatory patches. (Right) Saharwa type with dissemination. (Dr. Leon Goldman's case)

Associated lesions may be edema of the face and the ankles and erosion of the lips and the mouth.

The visceral lesions consist of a peculiar involvement of the endocardium (Libman Sacks syndrome) and polyserositis of the body cavities.

Systemic symptoms consist of high moderate or low fever delirium, chills, tachycardia and extreme fatigue

Recurrences often follow the use of various drugs as a result of extreme vascular sensitivity

Etiology The cause is unknown but the following theories have been advanced (1) primary vascular hypersensitivity to an unknown agent, (2) generalized bacterial allergy (tuberculosis or *Streptococcus haemolyticus*) and (3) a disturbance of the immune mechanism with the production of abnormal antibodies.*

The acute type occurs especially in young women between 15 and 30 years of age. The chronic type may be transformed into the acute type following a severe sunburn or injections of various drugs

See Gold, S. C. and Gowing, N. F. C. Lupus erythematosus, a clinical and pathological study Quart J Med 22 457 1953

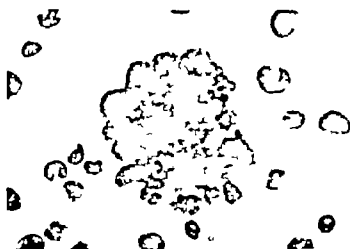


FIG 12 The L. E. cell found in the peripheral blood and bone marrow of acute systemic lupus erythematosus. The amorphous mass of nucleoprotein is surrounded by peripheral reaction of polymorphonuclear leukocytes (W. Landau South M J 46:3 11 1100, 1953)

Laboratory Data. The examination of the blood reveals progressive anemia, a shift to the left leukopenia, increased sedimentation rate, hyperglobinemia with a reversal of the albumin-globulin ration. Examination of the sternal bone marrow and centrifuged specimens of the peripheral blood reveals a peculiar cell, known as the "L. E. cell," which is a polymorphonuclear leukocyte whose nucleus has undergone autolysis in the presence of a factor associated with the serum globulin.*

Examination of the urine frequently reveals albuminuria and microscopic hematuria. Blood Kahn or Wassermann reactions are usually falsely positive because of the increased globulin in the blood.

Pathology Examination of the skin shows (1) liquefaction degeneration of the basal layer (2) dilatation of the superficial capillaries and the lymphatics in the upper corium and (3) fibrinoid degeneration of the collagen bundles.

The involved viscera present specific toxic effects with fibrinoid degeneration of the collagen.

Differential Diagnosis. The disease must be differentiated from acute erythema multiforme, acute rheumatic fever, dermatomyositis and acute solar dermatitis by the clinical course of the disease, profound systemic symptoms and chiefly the finding of the "L. E. cell" in the peripheral blood or the bone marrow.

Prognosis. The prognosis is guarded. Remissions may occur spontaneously or following ACTH or hydrocortisone, but when irreversible visceral and nephritic changes are present death usually occurs within weeks or months.

Treatment. Acute cases should be hospitalized in a darkened room. ACTH or hydrocortisone should be administered as soon as the diagnosis is made with careful urine and blood studies. After the symptoms are controlled treatment should be continued with prednisolone. Supportive treatment includes blood transfusions and large doses of ascorbic acid and rutin. Calamine lotion may be used for the skin lesions.

PITYRIASIS ROSEA

Pityriasis rosea is an acute generalized disease characterized by a more or less symmetrical eruption of pink or salmon-colored round or oval superficial macules with faint yellow or chamol-

*See Hasterick, J. R. Blood factor in acute disseminated lupus erythematosus. Arch. Dermat. & Syph. 61:209, 1950.

colored centers. Because of its characteristic distribution, sometimes it is called the "neck to knees" disease.

Varieties. (1) Macular (2) follicular (3) papular (4) vesicular and (5) aberrant.

Clinical Symptoms. The acute type is ushered in with fever adenopathy loss of appetite and fleeting joint pains. After the third day the characteristic eruption appears on the trunk and

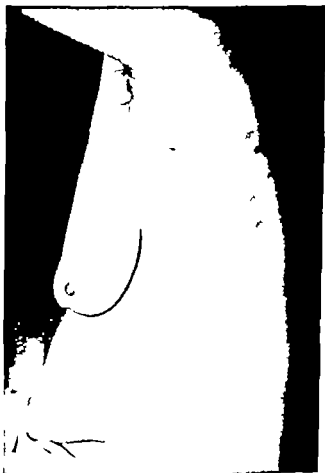


FIG. 13. Pityriasis rosea. Note large primary plaque.

spreads rapidly. The lesions in this type are apt to be more inflammatory than the common variety. The diagnosis may even remain in doubt for several days until the inflammation subsides.

Before the eruption appears, a primary herald initial or mother patch appears in about 60 per cent of the cases and precedes the generalized efflorescence. It is usually single but in rare cases may consist of two or more lesions. The primary patch, often mistaken for "ringworm," is larger and more inflammatory than the secondary lesions. It may appear on the chest, the abdomen or the thigh or, in fact, anywhere on the body.

Eruption After an incubation period of from 7 to 10 days, the general eruption appears, which consists of typical salmon-colored oval or round macules with yellowish crinkly centers. The long diameters of the lesions are parallel with the lines of cleavage. This is a diagnostic feature. The scales are limited to the margins of the macule and are very adherent, fine and non-greasy.

The eruption usually occurs on the trunk, the flanks, the upper chest and about the axillae. It is thickest on the sides of the trunk. The disease is uncommon on the face and the scalp, except in children and extensive cases, and rarely extends below the elbows or the knees. In rare cases erosive lesions may occur on the buccal mucosae.

Itching may be absent in the mild cases or it may be very severe. As a rule the more inflammatory the eruption and the more neurotic the patient, the greater the discomfort from itching.

The papular type is more common in Negroes and consists of acuminate papules interspersed with the ordinary lesions. In some cases the eruption tends to be follicular.

The vesicular type is rare and lasts for only a few days.

The aberrant type consists of a localized eruption limited to specific areas, including the arms, the axillary spaces or the thighs. Giant lesions are common in this variety.

In the Negro pityriasis rosea is apt to be more infiltrated, scaly and extensive. Even though the color markings are lost, the disease is not difficult to recognize because of the characteristic marginal scaling and the crinkly centers.

Etiology The disease appears in cycles with the peak in the spring and the fall although sporadic cases may occur during any month of the year. The condition affects the 20- to 40-year

old group especially. The nature of the disease is unknown. Suggested causes include (1) an external parasitic agent (*cryptococcus* of Dubois) and (2) a dermatropic hematogenous virus or organism which is too feeble to cause constitutional symptoms.

Course. The eruption persists for from 6 to 8 weeks or longer. Relapses are rare probably because the disease confers a certain amount of immunity. Temporary depigmentation may follow involution of the eruption after ultraviolet light therapy. Recrudescences or flare ups occasionally take place after the patient is well on the road to recovery. Fading of the erythema with increased scaling at the periphery of the lesions are signs of involution.

Diagnosis is based on the clinical characteristics of the lesions and the typical distribution of the disease along the lines of cleavage. In every case it is wise to examine the blood to rule out the presence of syphilis.

Pathology. The disease is characterized by a patchy parakeratosis slight acanthosis with intercellular and intracellular edema, tiny vesicles in the epidermis, moderate vascular dilatation and an infiltrate of polymorphonuclear leukocytes and lymphocytes in the upper corium.

Differential Diagnosis. PITYRIASIS ROSEALIKE ERUPTIONS include (1) drug eruptions following injections of bismuth or arsenic (2) dermatophytids (3) seborrheic dermatitis and (4) the acute phase of lichen planus. The disease must be differentiated from (1) secondary syphilis in which there are remains of a chancre possibly oral lesions, generalized adenopathy absence of marginal scaling and a positive blood test (2) seborrheic dermatitis in which there is localization in the mid-line of the chest and the back and flexural surfaces involvement of the scalp greasy scales and a chronic course (3) psoriasis in which the lesions are more infiltrated covered with silvery scales and tend to involve the extensor surfaces of the elbows and the knees, the scalp and the nails (4) disseminated tinea cruris in which there is a primary focus in the genitocrural region positive cultures and absence of localization along the line of cleavage.

Treatment. Although pityriasis rosea is a self limited disease it should be treated for psychologic reasons to hasten involution and to reduce the itching. Nothing but mild applications should be used. Lotions are preferable to ointments. A neocalamine and zinc oxide lotion with 1 per cent resorcinol applied twice daily until the lesions lose their pink color and start peeling is gen-

erally effective. If the skin becomes excessively dry from the use of the lotion light mineral oil or Nivea skin oil should be substituted for a few days. If lotions are not tolerated, 2 per cent salicylic acid in talcum powder is effective. Ultraviolet radiation at 5-day intervals in doses to cause slight erythema hastens recovery.

Fractional roentgen ray therapy by an experienced dermatologist is useful in the papular types to hasten involution and to diminish the pruritus. Hot baths should be avoided.

NURSING ASPECTS

Careful examination of the entire skin and the mucous membranes is necessary in arriving at a diagnosis in diseases of the erythema group. The patient's temperature should be taken before the physician comes into the room. Facilities should be available for blood examinations.

Bed Sores. Nursing care is important to help to overcome circulatory resistance over pressure areas. A clean bed smooth linen neat dressings, frequent changes in position massage and alcohol rubs, if ordered are important. A high-vitamin, high-caloric diet daily inspection of the entire skin for dull-red areas (early lesions) air mattresses, invalid rings and the generous use of talc powder are routine procedures.

Urticaria. The patient should be advised to avoid wearing constricting articles of clothing. Epinephrine solutions should be available, as well as injectable antihistaminics. The nurse should instruct the patient in the use of the colloid bath.

Serum Sickness. Epinephrine solutions and ACTH gel should be available.

Acute Lupus Erythematosus. Since patients with this serious disease usually are hospitalized special care is necessary. Complete bed rest is important because of the frequency of cardiac complications. The temperature and the blood pressure should be recorded at regular intervals. The room should be darkened because of the possibility that light sensitization will affect the extent of the eruption. Since ACTH or Hydrocortisone is the treatment of first choice apparatus for giving the drug by intravenous drip or intramuscular injection should be available. Reactions from the drug (hypertension, impaired renal function, impaired cardiac reserve, abdominal pain, personality disorders or mental confusion and edema) must be kept in mind constantly. Materials should be available for the discovery of the "L.E." cell in the blood or the bone marrow.

The Eczema Group

ATOPIC ECZEMA	CONTACT DERMATITIS
INFANTILE ECZEMA	PLANT DERMATITIS
INFECTIOUS ECZEMATOID DERMATITIS	IVY POISONING
VARICOSE DERMATITIS	OCCUPATIONAL (INDUSTRIAL) DERMATOSES
	NUMMULAR ECZEMA
	NURSING ASPECTS

ECZEMATOUS dermatitis is an acute subacute or chronic polymorphous eruption in a predisposed individual. It is characterized by (1) three progressive clinical stages consisting of erythema, exudation or edema and infiltration (2) the presence of itching or burning sensations and (3) hypersensitivity to improper local medication. The exciting factor may be trivial, but the predisposing factors, which vary in each individual, are responsible for the epidermal and biologic response.

The term *eczema* in a strictly limited sense, is used to describe local or generalized patches of dermatitis of internal (endogenous) origin. Eczema is not a complete scientific diagnosis but a descriptive term for a clinical reaction. If the exciting factor is known a qualifying adjective is added to denote the cause e.g. nutritional atopic, gravitational etc.

Dermatitis however is an inflammation of the skin caused by external (exogenous) chemical or physical irritants or sensitizers. Contact dermatitis is a form of allergic external dermatitis. The term *dermatitis venenata* has a limited application (dermatitis caused by plants and plant products).

An eczematous skin is hypersensitive to mild irritants such as soap water wool etc. to trauma from scratching or friction and to temperature changes. It is subject to increased itching from tension worry and fatigue. It is characteristically intolerant to the wrong application or to the right one used at the wrong time*.

*For a valuable discussion of the practical and theoretical aspects of the subject, consult Loewenthal, L. J. A. Ed. *The Eczemas: A Symposium* by Ten Authors, Livingstone, London, 1954.

Since many chronic eczemas have a multiple etiology each contributing factor must be determined evaluated and eliminated if possible. An interesting practical review of the pathogenesis of the eczemas by Pillsbury Milescher Brain, *et al* will be found in the Proceedings of the Tenth International Congress of Dermatology London 1952 pages 3 to 126

Eczema can be prolonged and made resistant to therapy by the use of irritating preparations overtreatment, overdosage with x-ray therapy and various exogenous and endogenous causes.

Pathology of Eczema. Since eczema is an unstable and polymorphous condition the pathology varies with the stage of the disease. The main characteristics are.

ACUTE CASES. The stratum corneum is replaced by crusts of dried exudate. Exposure of the rete is due to destruction of the stratum corneum by edema. Intracellular and intercellular edema of the prickle-cell layer results in vesicle formation in the upper corium and the epidermis. Vascular dilatation, a moderate perivascular round-cell infiltration and edema are present in the corium.

CHRONIC CASES. Depending on the stage, hyperkeratosis and parakeratosis (scaling) spongiosis (edema of rete) and acanthosis (elongation of rete pegs) are characteristic findings. Perivascular infiltration consisting of round cells, polymorphonuclear leukocytes and mast cells is found in the corium

ATOPIC ECZEMA

Atopic eczema is an acute, subacute or chronic relapsing pruritic dermatitis in predisposed individuals and is characterized by a selective localization and extreme chronicity. The secondary changes in the skin (eczematization lichenification) are the results of scratching or rubbing extremely irritable and pruritic areas. A psychosomatic factor may fatigue the higher cortical centers interfering with normal inhibition of the scratch reflex.

Clinical Characteristics. The first stage of the disease often begins soon after birth as a pruritus that is followed soon by an acute or subacute erythematous or weeping pruritic dermatitis on the face and may become more or less generalized. After the second year the eruption tends to disappear but may recur as the second stage, which begins early in life at or before puberty as a localized intense pruritus of the flexure surfaces of the elbows the knees, the neck or the face. The resulting eczematoid areas as-

sume the characteristics of an acute subacute or chronic dermatitis, depending on the time element. The dermatitis is ill-defined, scaly thickened and covered with blood crusts or excoriations from scratching. In the chronic lichenified types there is thickening of the epidermis, exaggeration of the normal creases of the skin and extreme dryness.

Itching becomes a conditioned reflex and any emotional upset can bring about an uncontrollable attack of pruritus.

THE SITES usually involved are the anterior cubital fossae the popliteal spaces, the sides and the back of the neck and the face.

Etiology THE PREDISPOSING FACTORS include a basic hereditary hypersensitivity an abnormal tissue response to scratching vasomotor instability sweat retention syndrome (plugging of the sweat pores resulting in itching) and a dry skin (xeroderma)



FIG. 14 Atopic eczema of the cubital spaces.

THE EXCITING CAUSE is frequently unknown or it may be a primary irritant hyperhidrosis (summer flare ups) physical or mental exertion or an emotional upset.

The Selye alarm reaction may explain some cases, but much remains unknown concerning the pathogenesis.

Foods may be the trigger mechanism in about 15 to 20 per cent of the cases in infants. Attempts to find the cause of atopic eczema are made difficult by the fact that most patients are sensitive to more than one allergen (polysensitivity) and we are ignorant of the real cause of the condition.

Diagnosis. The localization on the anterior cubital fossae and the popliteal spaces is characteristic. Supportive evidence includes the history of beginning in childhood a hereditary allergic background severe pruritic crises extreme irritability of the skin frequently improvement during the warmer months and extreme chronicity.

CUTANEOUS TESTS The wheal or flare response from intra-dermal or scratch tests using animal or food proteins is of no value in diagnosis, and an unnecessary expense because experience has shown that results are misleading and unreliable.

PSYCHIATRIC EXAMINATIONS A study of the home environment and psychosomatic factors is frequently of value in the determination of the cause of flare-ups.

ELIMINATION DIETS may provide useful information in conjunction with a study of the history in children. They are best carried out in the hospital. These detailed menus must be adequate in calories and vitamins as well as nutritious and satisfying.

Differential Diagnosis. **CONTACT DERMATITIS** is an acquired sensitivity due to exogenous allergens. At first the eruption is limited to the contact site. Autosensitization (spreading factor) may develop as a complication with involvement of the same areas affected by atopic eczema.

SEBORRHEIC DERMATITIS involves the flexures, but the greasy scaly patches are ill-defined and less infiltrated than atopic dermatitis. The disease is also present in the scalp and usually affects the mid-line of the face.

Course and Prognosis. The disease is characterized by remission and exacerbations of various intensities and extent. These continue at indefinite intervals throughout childhood and adult life. While most cases improve during the warm months, the sum-

mer type tends to become more acute and eczematoid dermatitis on the trunk or extremities appears and pruritus becomes more pronounced. In children the antecubital spaces may be the only site affected but the eczema may become generalized at any time. In people past 40 the affection tends to become more or less quiescent with the passing years. Trauma from scratching and intolerance to local therapy are factors that often prolong attacks. Those cases associated with asthma are intractable. Steroid therapy has improved the general outlook.

Treatment. The management of this disease frequently taxes the ingenuity of the physician. If the disease is extensive hospitalization should be advised some cases improve considerably with even one week of complete bed rest. This measure results in temporary improvement by removing the patient from his contacts and freeing him from his business and domestic worries. In intractable cases a geographic change from a cold area to a sunny warm dry section of the country may benefit, at least temporarily, those definitely bothered by cold weather those helped by exposure to mild sunshine and those with respiratory complications (Smith and Garrett).

LOCAL TREATMENT The skin is extremely irritable so that only bland ointments should be used until the skin tolerance is determined. Soap and water antihistaminic ointments and even a calamine lotion may precipitate an attack during the reactive phase of the disease. For cleansing a light mineral oil cold cream, or sulfonated oil should be used. In the acute cases compresses of boric acid or 1:20 Burow's solution are useful. Sub-acute cases may respond to 1 per cent hydrocortone in Acid Mantle Cream (Dome). The chronic cases should be treated with tar ointments of graduated strengths beginning with 3 per cent Dernaftan combined with $\frac{1}{4}$ per cent hydrocortone in hydrophilic ointment—U. S. I. When using these ointments the patient should be under close observation so that intolerance can be detected as soon as possible and the ointment discontinued.

When the eczema is limited to small areas, hydrocortone ointment is useful but when it is discontinued the eruption usually recurs. Antihistaminic systemic therapy is also prescribed.

In quiescent cases in which extreme dryness associated with cyclic pruritus are the only complaints infrequent bathing avoidance of wool and exposure to sudden changes in temperature and

the use of Dumolene or Nivea skin oil for lubrication may give symptomatic relief.

NONSPECIFIC DESSENSITIZATION may be attempted by the following measures: pyromena, calcium (Calcibronat—Sandoz) or strontium bromide, or autohemotherapy.

Antihistaminic drugs are valuable as sedatives in about 30 per cent of the cases, but whether they control the pruritus by protecting the skin from the effects of free excess histamine released under pathologic conditions is a controversial question.

In those cases with a definite psychosomatic background consultation with an understanding psychiatrist may be beneficial.

ACTH, Cortrophin-Zinc, and cortisone should be reserved for the generalized cases. They afford temporary relief. Sedation in acute cases may be obtained with a trial of the various barbiturates, chloral hydrate or paraldehyde.

Patients with atopic eczema should not be exposed to herpes simplex because of the danger of a superimposed Kaposi's varicelliform eruption, and to recently vaccinated persons because of the danger of eczema vaccinatum.

Instructions For Patients with Atopic Eczema

The Cause. Regard your condition as you would a birthmark. Learn to live with it and learn how to care for it. It is only a type of itching that is transferred into an eczema by your scratching.

Mental Outlook. Do not let it depress you. It is not a serious condition—only bothersome. Anger, resentment and overtiredness will increase the itching.

Bathing. Since the skin is dry and irritable, a tub bath every other day in winter using Aveeno or Basis soap is more agreeable. Blot—do not rub with the towel. It is useful to apply Nivea skin oil to the dry thickened areas after the bath.

Clothing. Wool shirts, sweaters or underwear irritate the skin and cause itching.

Temperature. A sudden change like dressing in a cool room or facing cold winds will start up a spell of itching. Too many covers on the bed heat up the skin and cause itching.

Itching. This is always worse at night because unconscious scratching during your sleep interferes with the healing process. Keep your arms covered with tubular gauze and take the night pills ordered for you. Keep your fingernails short or wear thin cotton gloves ordered for you.

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ally milk, eggs, wheat, chocolate or cod liver oil and overdosage with vitamin A may aggravate the eruption

Prognosis This type usually clears by the second year

Treatment Mild cases may be treated at home. Severe cases are best treated at the hospital where dietary supervision may be obtained proper restraints to avoid scratching used, and therapeutic results evaluated.

Pruritus in infantile eczema is not relieved by oral antihistamines but responds best to Dulcets Aspirin gr 1 in my experience

The acute cases are treated with continuous wet packs of aluminum acetate or 1/6,000 permanganate. Crusts are removed with warm olive oil or starch paste. Cleanliness is important. The subacute types often respond to Cortidome or Lassar paste containing 2 per cent Xeroform. The chronic types are treated with ointments containing Dernaftan (Vacro Products) or crude coal tar in increasing strengths [Daxalan (Dome) Zetar (Der milk) or Co-Ga Tar (Texas Pharmacal)] to which is added 1/5 per cent hydrocortisone.

The diet should be normal for the age of the infant. A daily weight chart should be kept.

2 Atopic Type. These cases are the most difficult of all to treat. The condition, which is the first phase of disseminated neurodermatitis is characterized by (1) a restless, underweight irritable infant (2) extreme pruritus (3) a dry or moist eczema told dermatitis of the face, the anterior neck, the flexures of the elbows, antecubital spaces and the dorsum of the hands (4) anxious parents, worn out by lack sleep and (5) intolerance to all but the mildest of local applications. Teething colds, digestive disturbances sudden changes of temperature and "temper tantrums" may cause flare-ups

Complications include impetiginization, secondary eczematization Kaposi's varicelliform eruption and, rarely acute glomerulonephritis from absorption of phenol or ammoniated mercury preparations used in the treatment.

Etiology These children are born with a sensitive skin. The importance of skin testing in these cases has been overstressed because removal of reacting foods or epidermals, as determined by testing does not alleviate the eruption. Identification of the causative allergen is often difficult but the history may give valuable information.

Prognosis is guarded. About 10 per cent persist as neurodermatitis of childhood and 15 per cent as adult neurodermatitis.

Prophylaxis When there is a family history of allergies, this type of eczema may be prevented in some cases by adherence to the following (1) introduce one simple food at a time at 5-day intervals (2) avoid all complex foods and cereals until the fifth month (3) give no eggs the first year (4) avoid commercial baby oils, overcleanliness and the use of detergents in the infant's laundry

Treatment requires patience and skill. Hospitalization is important. Restraints, sedation elimination diets, evaluation of therapy and "therapeutic neglect" are best carried out in the hospital by a trained personnel. The evaluation of the history and the clinical results of elimination diets may suggest milk, eggs, wheat, orange juice and other foods as a contributing cause. The food under suspicion should be eliminated for at least three weeks to have value as a therapeutic test. In any case the baby should not be permitted to lose weight or to develop hypoproteinemia or iron deficiencies. Immunization injections should be withheld until summer. ACTH should be reserved for the persistent severe cases if hospitalized, while ambulatory cases often respond to 5 mg. hydrocortisone (12 drops of wild cherry syrup equals 5 mg.) given 4 or 5 times daily. After improvement the eczema may be controlled with a maintenance dose of 5 mg. daily. Steroid therapy does not replace conventional local therapy.

Dietary control should be placed in the hands of the consulting pediatrician who knows more about baby feeding than the average dermatologist. Some experienced investigators claim that evaporated milk is of little value that orange juice seldom is allergenic and that boiling milk or eggs has a dubious effect on their allergenicity.

If milk appears to be a factor then a trial of soybean substitutes (Mull Soy Sobee Nutramigen) is indicated. If the infant is over 6 months of age, and has learned to like the taste of cow's milk, the chances are that he will refuse these products. At this stage canned goat's milk may be introduced for better or for worse.

Local therapy Baby oils, antihistaminic and antibiotic ointments usually aggravate the condition. The following ointments are worthy of a trial: hydrocortisone ointment, pediatric Davalene or Cortodone ointment. These should be removed with sterile olive or mineral oil or plain Phisoderm.



FIG. 15 Infectious eczematoid dermatitis in pediculosis capitis.

3 INFECTIOUS ECZEMATOID TYPE This type is usually secondary to an external infection often a rhinitis, a running ear neglected impetigo or poor skin hygiene. The eruption is primarily vesicular but soon becomes crusted, pustular and later eczematoid. The staphylococcus or the streptococcus are the causative organisms.

Prognosis is good since with proper therapy the average case clears up within two weeks.

Treatment Bacitracin ointment and neomycin ointment often are effective and rarely sensitize. If response is slow Terramycin pediatric drops (Pfizer) or dulcet Penicillin G Potassium tablets (Abbott) every 3 hours for 3 or 4 days may be of value.

4 XERODERMATIC TYPE. Infants with this form of winter eczema are born with a dry skin, lacking sweat and oil glands. The entire skin but especially the face and the flexures, are

easily irritated by soap and water cold weather wool and friction Pruritus is moderate. The condition clears with the onset of warmer weather

Treatment consists of infrequent bathing liquid petroleum for cleansing and the use of vitamin A or lanolin ointments. Small doses of thyroïd may be indicated

5 SEBORRHEIC TYPE. Although, theoretically seborrhea can not occur until puberty the scalp the ears and the so-called "seborrheic areas" may be affected by a greasy scaly dry or eczematoid patchy eruption. In some cases the infection is acquired from the nurse or the parents.

Treatment consists of 2 per cent colloidal sulfur or ammoniated mercury ointments, B vitamins and dietary regulation. A low fat diet including skimmed or evaporated milk or Dryco (Borden) may be useful Neocortef ointment is useful in stubborn types

General Directions for Infantile Atopic Eczema

1 Be calm and patient. Nervousness on your part only serves to make the child more restless and aggravates the itching

2 Handle the child as little as possible.

3 The child's bedroom should be free of "allergic" materials. No rugs, curtains plants, birds, cats or dogs should be permitted. Do not use woolen blankets. Mattress and pillows should be stuffed with kapok (no feathers)

4 The room should be dusted only when child is elsewhere

5 Do not use bleaches on bed sheets and pillow cases. Use three rinses on these articles to get all soap out of the material.

6 No soap should be permitted on eczematous areas. Special soap will be ordered for cleanliness, and cleansing cream for itching areas.

7 If the child scratches or rubs eczematous areas, restraints should be ordered

8 You will be given several ointments to try as no single ointment works the same in all cases.

9 The diet should be changed only if and when local treatment fails to bring results.

LOCALIZED NEURODERMATITIS

Localized neurodermatitis (lichen simplex chronicus) (see p. 261)

INFECTIOUS ECZEMATOID DERMATITIS

This is a dermatitis occurring near the site of a discharging surface or an external focus of infection. The condition results from a local sensitization to bacteria or other products in the exudate or the secretion. The eruption may be follicular eczema, toid, papular vesicular or pustular. The acute types are characterized by local edema, crusting, weeping and intense itching. The chronic types are usually pustulovesicular and infiltrated. Any of the types may become generalized by absorption of the products of inflammation and subsequent auto sensitization.



FIG 16 Infectious eczematoid dermatitis of the ear cured with hot boric packs and Bactracin Ointment

Etiology The condition may occur about infected wounds, in rhinitis, purulent conjunctivitis or sinusitis, vaginitis, proctitis, otitis media osteomyelitis, varicose dermatitis furuncles or ulcers. It is often a complication of seborrheic dermatitis, infantile dermatitis, intertrigo and contact dermatitis of the hands resulting from the use of strong local applications. Scratching or rubbing with subsequent inoculation of staphylococci streptococci or colon bacilli on a localized allergic skin initiates the disturbance.

Differential diagnosis is from contact dermatitis and impetigo.

Treatment. Cultures should be taken to determine the type of bacterial infection. Penicillin injections (Duracillin is preferred as less sensitizing) up to 3 million units are useful in some cases, unless patient is sensitive. Localized infections if not on the exposed areas usually respond to daily paintings of 2 per cent aqueous gentian violet and bacitracin ointment or neomycin ointment. There is doubt in my mind that a mixture of antibiotics is more effective than a single agent.

Acute cases should be treated with hot boric packs or tyrothrycin wet dressings and Terramycin or Aureomycin (250 mg 4 times daily for 3 or 4 days) Pruritus if present, should be controlled with oral antihistaminics (Benadryl or Phenergan) rather than antipruritic ointments which often are sensitizers.

VARICOSE DERMATITIS

Varicose dermatitis (dermatitis hemostatica) is part of the varicose-vein complex. The condition begins with gravitational congestion followed by edema and a bluish-red hyperemia. The itching which is usually intense, results in excoriations and a traumatic dermatitis. Absorption of the products of exudation results in auto sensitization or secondary eczematization or both. The condition is due to a lowered resistance of the parts from lack of oxygen and poor venous return. Varicose dermatitis is apt to be persistent unless the causative factors are eliminated. Secondary infection localized neurodermatitis, ulceration, thrombophlebitis and lymphedema are common complications.

Treatment of the acute cases should consist of hospitalization complete bed rest, elevation of the legs, continuous wet astringent compresses oral terramycin or achromycin for secondary infection regulation of the contributing or aggravating factors and local soothing therapy. In the subacute types (moist eczematoid dermatitis) calamine liniment packs during the day and a 5 per cent bismuth tribromphenate in Lassar's paste at night with support of an elastic bandage and mild antihistaminics to control pruritus are useful. In the dry infiltrated chronic types the following ointment, if tolerated has given valuable service.

Dermastan	5
Hydrocortone	0.5
Lassar's paste	
Hydrophobic Ointment—U.S.P. 11 ad 100.5	Apply as
mosks twice daily	



FIG 17 A typical case of dye dermatitis caused by wearing a black dress. Note the absence of involvement of the axillary vault.

High subcutaneous ligation to prevent recurrence, is necessary after the dermatitis is controlled.

CONTACT DERMATITIS

Contact dermatitis (*dermatitis venenata*, *eczematoid dermatitis*) is an acquired epidermal sensitivity to continuous or intermittent external contact with a vegetable mineral or animal substance over a variable period of time. These irritants consist of a wide variety of substances, including chemicals, soaps, plants, drugs, dyes, pollens and various products used in the home and in industry.*

*For an extensive discussion with illustrations of special sites affected by various contactants see Waldbett, G. L., *Contact Dermatitis*, Springfield, Ill. Thomas, 1953.



FIG. 18 Contact dermatitis from gasoline.

Clinical Considerations. The cutaneous reaction varies from slight itching to severe vesiculation. The distribution of the eruption which is of diagnostic importance at first naturally involves only those areas exposed to the irritant. After several days or weeks the eruption may spread to other parts of the body by a reaction of the tissues called autosensitization. Three definite periods occur following exposure to a reactive substance (1) tolerance the asymptomatic stage during which the substance is in contact with the skin (2) sensitization the stage during which the skin becomes sensitized and (3) reaction the stage characterized by the sudden appearance of the eruption.

Contact dermatitis consists of the following characteristics (1) an eruption developing in from 12 hours to 3 days after the last exposure (2) erythematovesicular in its early stages (3) indefinite margins (4) a reaction spreading beyond the limits of the original exposure (5) a definite history of exposure (6) unusual site as compared with orthodox eruptions.

In the beginning the patient is often sensitive to only one specific contactant. In some cases, however, this specific sensitivity may be followed by a low-grade nonspecific sensitivity to other related or unrelated substances (polysensitivity). When this occurs, recovery is naturally delayed because of the recurrent or prolonged cutaneous reaction.

Diagnosis is made by (1) noting the site of the eruption (2) taking a detailed history (3) limitation of the dermatitis to a specific area in most cases, (4) a knowledge of the possible contactants that could cause a dermatitis in that area and (5) the use of the patch test.



FIG 19 Contact dermatitis from black-dyed shoe leather. Often, these cases are diagnosed as "athlete's foot" and usually are aggravated by fungicidal ointments.



FIG 20 Contact type of eczematous dermatitis resulting from sensitization to black shoe polish

THE PATCH TEST This diagnostic procedure when positive results in the production of a miniature contact dermatitis. It is made directly on the skin reproducing the natural factors responsible for the eruption. The test site must be on normal skin as close to the eruption as possible. For contact dermatitis of the face, the "V" of the neck is employed for the test.

If dry substances, such as powders, are employed they are first moistened with distilled water and placed on a square of linen about half the size of a postage stamp. This is then at



FIG. 21 Acute contact dermatitis from sensitization to *sulfis* thiarsole ointment (From Dr Harry Yoslik)

CONTACT SURVEY

In order to determine what is irritating your skin, please list all substances, materials, cleansers and products which you use, wear, touch or handle and which come in contact with the skin of your hands. You will be given seven sheets, one for each day. Be thorough and observant.

	ACTIVITY AT THE TIME	SUBSTANCES HANDLED	REACTED IN REACTION FOR ANY 24 HOURS (APPEARANCE AND RECORD)
AM			
6			
7			
8			
9			
10			
11			
12			
PM			
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			

FIG. 22 This outline is used in cases of contact dermatitis in which the cause is not apparent.

tached to the skin with Elastopatch (Duke) or flexible collodion. The patch is left on the skin for 24 hours or until itching or dermatitis is noticed, indicating a positive reaction. Liquids are applied to blotting paper and the same technic is followed. If the substance is a leaf or similar vegetable matter it is slightly macerated and placed directly on the skin. Volatile substances are applied to the skin for 15 minutes using an inverted test tube to prevent evaporation. The reaction to a properly applied

patch test depends on (1) the degree of the patient's sensitivity (2) the concentration of the substance applied * (3) the amount of substance applied per sq. cm. of skin and (4) the amount of time that it remains in contact with the skin.

Reading The test is read at the end of 24 hours and also at the end of 48 and 72 hours (delayed reaction). The results are indicated as follows

Itching	1 plus	Papules	3 plus
Redness	2 plus	Vesicles	4 plus

Occasionally specific reactions cause a flare-up of the original eruption. Nonspecific positive tests may result from the use of adhesive plaster strong concentrations of the irritant, heavy metals and pilosebaceous irritants.



FIG. 23. Housewife's eczema, common in young mothers, contact type of eczematous dermatitis resulting from sensitization to soap.

Reporting the patch test This should show the substance used the concentration the site the solvent the amount used the size of the area (3 sq. cm.) the length of time and the result in plus signs.

DANGERS OF PATCH TESTING include (1) local necrosis from using primary irritants (2) generalization of a previously localized eruption (3) sensitization of a previously normal skin and (4) constitutional allergic symptoms.

For concentrations and vehicles used in patch testing see *Text Book of Dermatology and Syphilis* by 1943 pp. 25-42.

Procedures If Cause Is Unknown. Special investigation is necessary. When a flare up occurs in a chronic dermatitis the sensitizer may be discovered by asking the patient what he was doing wearing or using within 24 hours of the recurrence. Patch tests are then made with all the suspected substances. The choice of these tests depends on the exposure possibilities. Indirect contacts, e.g., dermatitis in a husband from contact with his wife's hair tonic, dermatitis on the arm from contact with an ointment used on the scalp also must be checked. An hour-to-hour diary listing all possible direct and indirect contacts within a 24-hour period may provide important information not otherwise obtainable.

Etiology A very common dermatosis allergic contact dermatitis, is caused by any agent (contactant) which may come into

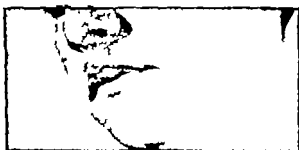


FIG. 24. Contact dermatitis of the upper lip (sensitization to penicillin ointment)

repeated contact with the skin or the mucous membranes. During the latent period which follows the first contact, the exposed area becomes sensitized to the offending agent. Common contactants include (1) soaps and detergents, (2) drugs incorporated in ointments lotions etc. (penicillin, sulfonamides, tar Menthio-late Nupercainol sulfur anesthetics, phenol furacin antihistamines, resorcinol ammoniated mercury etc.) (3) cosmetics (4) dyed clothing and footwear (5) jewelry and (6) plants.

Localization and Possible Causes of Contact Dermatitis

General: Woods, plants, local applications

Scalp: Hair tonics and dyes, lacquered hair pins

Forehead: Hat bands, scalp pomades and lotions

Ears: Telephone receiver spectacles (white gold or tortoise-shell) perfume

Eyebrows: Cosmetics

- Eyelids Cosmetics, eyelash curlers, manicure preparations (indirect)
 Lips: Lipstick, dentifrice, glue foods (orange juice, tomatoes, spinach, etc.)
 diethylene glycol (cigarettes)
 Face: Cosmetics, rubber gas masks, plants, insecticides, soap, scalp preparations, rubber sponges
 Neck Necklaces, fur dyes, perfume and nail polishes
 Arrière: Depilatories, astringents, dress shields, dyes
 Chest Adhesive plaster liniments, wool and silk
 Back: Adhesive plaster, medicated plasters, liniments, wool and silk
 Genitocrural Region Parasiticides, urine, trusses
 Genitals Parasiticides, antiseptic washes and douches, tincture of iodine, rubber condoms
 Buttocks Weeds and grasses, toilet seats
 Anal Region Suppositories, antipruritic ointments and "pills" remedies, nail polish
 Thighs Match boxes
 Legs Plants and weeds, leather puttees, wool, soap
 Feet Leather and dyed shoes, socks and bedroom slippers, and Whitfield's ointment
 Arms Soaps, dyes, wool
 Forearms Oils and greases
 Wrists Watch straps, bracelets, leather or dyed gloves, wristwatches
 Hands Soap, water polishes, detergents, cleaning powders, paint and paint removers, drugs, procaine antiseptics, acetone, formalin, glue etc.
 Fingers Manicure preparations, carbon paper finger paints

Contact dermatitis in children is infrequent but may be caused by soap dyed clothing wool garments, painted toys, varnished furniture linoleum, carpets and furniture polish.

Prognosis is good if the cause is found and removed.

Treatment. (1) Remove the irritant from the environment of the individual (2) discontinue all previous therapy and (3) apply the mildest of local applications indicated. In the acute types (hyperemia or vesiculation) cool wet packs of 1 per cent aluminum acetate or calamine lotion or a zinc oxide-corn starch shake lotion (p. 602) for several days are indicated for the subacute types (erythema with scaling or crusting) calamine liniment or Lassar's paste is useful and for the chronic types (dry lichenified areas) hydrocortisone ointments or x ray therapy may be effective. The affected areas should be dressed twice daily for protection from irritation by the clothing and to avoid secondary infection. If pruritus is present, a mild antihistamine (e.g. Neoantergan) may be ordered for sedation.

If the eruption persists in spite of removal of contacts, all treatment should be discontinued and the case reviewed from the standpoint of diagnosis and sensitization to all drugs, ointment bases and injection therapy as well as psychosomatic factors.

Prophylaxis. When the cause is known, the contactant should be avoided to prevent further attacks. If the dermatitis is caused by sensitivity to local medication the patient should be informed of the fact and instructed to advise physicians she might consult at some future time. Cotton-lined rubber gloves should be worn by housewives in cases of contact dermatitis of the hands from detergents and other products used in the home.

Silicone ointments are useful skin protectants for persons unavoidably exposed to detergents and other external irritants. The number of applications depends on the amount of exposures. The ointment should not be used on weeping infected surfaces.

PLANT AND WEED DERMATITIS

Plant dermatitis is usually a seasonal dermatitis, affecting the exposed surfaces in predisposed individuals.

Clinical Signs. The eruption which is acute and vesicular in the beginning appears on the face the hands, the legs and the genitals. It usually is associated with marked burning and itching. As in other types of contact dermatitis, the inflammation spreads to other parts of the body by scratching, by direct transfer or by the entrance of the allergen through light, porous clothing. The dermatitis may last for several weeks depending on the degree of sensitivity. Secondary infection may set in long after the exciting cause has disappeared because of the lowered resistance of the skin.

Etiology The disease affects amateur and professional gardeners, game hunters, hikers, farmers ranchers and those whose work or pleasure places them in contact with plants. Indirect contact may occur through the medium of cows, insects, clothing, drugs and smoke. The following includes the usual weeds which produce contact dermatitis

Bittersweet
Brodia weed
Burweed
Cocklebur
Gaillardia (Indian)
Marsh Elder

Pasture Sage
Ragweed, Giant
Ragweed, (Dwarf) Short
Ragweed, Western
Sawtooth
Timothy

POLLENS GRASSES AND WEEDS occasionally affect the skin during the growing period of the plants. The eruption usually occurs on the face but stops at the collar line. In extensive cases the neck, the hands, the ankles, the knees and the genitals may be

affected. There is usually a history of seasonal incidence. The eruption disappears after the first killing frost and returns in the early summer.

Diagnosis is made by patch tests using the resins of the plants, and also by the history.

Differential Diagnosis. Some cases may be mistaken for atopic eczema but the seasonal incidence and the manner of evolution should help to avoid confusion.

Prophylaxis. Preseasonal desensitization with the pollen oils is usually unsatisfactory. The patient should wear long underwear or puttees to protect the skin and should avoid the suspected plants so far as possible. In most cases a change of occupation is the only measure which prevents a return of the eruption.

Prognosis. The dermatitis continues with exacerbations and remissions all through the season until the exciting cause is discovered and removed. The eruption is, of course, more severe at the original sites of exposure.

Treatment consists of avoidance of the plant, oral desensitization * intramuscular injections of specific vegetable-oil extracts, soothing lotions or mild ointments, as indicated and a trial of the antihistaminics for relief of the pruritus.

IVY POISONING

Ivy poisoning (*Dermatitis venenata*) is a common form of plant dermatitis in the United States and is characterized by a severe vesicular dermatitis on the legs, the flexure surfaces of the arms and the forearms, the dorsum of the hands and the interdigital spaces, the genitals and more or less edema of the face. The incubation period varies from 12 hours to 7 days. In the Midwest cases are seen as early as March.

Etiology. The plants which cause ivy poisoning are closely related and belong to the genus *Rhus*. There are three poisonous species: (1) poison ivy (*R. toxicodendron*) (2) poison oak of which there are two varieties, *R. quercifolia* of the Eastern States and *R. diversiloba* of the Pacific States, (3) poison sumac found about swamps. In order to acquire the dermatitis, which is a matter of individual susceptibility, it is necessary to come into direct contact with the sap or the resin of the plant, its leaves or

* For dosage consult booklet, *Oral Desensitization in Plant Dermatitis*, Graham Labs, 6146 Willow Lane, Dallas, Tex.



FIG. 25 Poison sumac (*Toxicodendron vernix* [L.] Kuntze.) (From a water color by F. A. Walpole reproduced Farmer's Bulletin 1166, U. S. Dept. of Agriculture)

berries. The active chemical principle common to the group is urushiol.

Prophylaxis. Immediately after exposure and before the eruption appears, the patient should take a hot bath with laundry soap. The nails should be cleaned with an orange-wood stick to remove any resin that remains in that area. The entire body then should be sponged with benzine for several minutes fol-

lowed by a cleansing soap bath. All the clothing worn at the time of exposure should be dry cleaned. Zirconium hydrate ointment if used within 1 hour of contact is said to inactivate urushiol by precipitation (Cronk) Further confirmation is necessary

Treatment. Ointments or oils at the onset of the eruption are definitely contraindicated, since the irritating principle is oil soluble. If this fact is ignored there is danger of spreading the eruption. In the beginning open wet packs of 1/5,000 potassium permanganate often will reduce the edema and the vesiculation. The following prescription is also valuable as a soothing and astringent application

Phenol, liquefied	10
Zinc oxide	12.0
Lime water q.s. ad	100.0

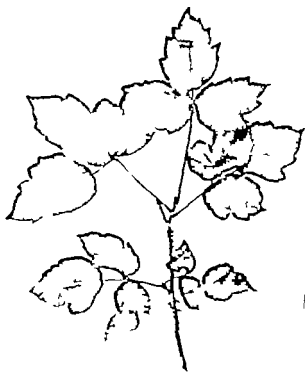


FIG. 26 Poison ivy, *Rhus ex.*

If bullae are present, they should be opened aseptically and painted with 5 per cent potassium permanganate solution or a lotion containing tannic acid (e.g., Rhullitol) should be applied for not longer than 2 days.

CORTICOTROPINS. Severe cases may be controlled with ACTH or cortisone if not contraindicated for not longer than 3 or 4 days with gradual reduction of the dose to avoid the rebound phenomenon. Hydrocortone lotion is useful for localized areas.

ANTIGEN INJECTIONS should be avoided in all cases since auto-sensitization may occur and prolong the disease.

PRURITUS. This is best controlled with colloid baths (starch, baking soda, Aveeno) and mild antihistaminics.

PREEASONAL DESSENSITIZATION. There is no adequate proof of the effectiveness of measures directed toward desensitization.

Oral prophylaxis with increasing doses of the oleoresin (1.25) is useful in highly sensitive persons in spite of the short period of immunity (about 4 months) and the high percentage of reac

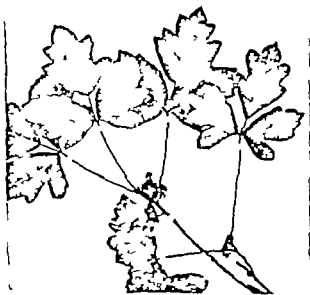


FIG. 27 Poison oak leaves (*Toxicodendron quercifolium* [Michx.] Greene) of the eastern United States. (Farmer's Bulletin 1166, U. S. Dept. of Agriculture)



FIG. 28 Contact dermatitis—concrete worker (U S Public Health Service Office of Dermatoses Investigations)

tions (25 per cent). Probably the best means of prophylaxis in those whose occupation exposes them to poison ivy is the use of silicone ointments and protective clothing.

OCCUPATIONAL (INDUSTRIAL) DERMATOSES

This subject has assumed much importance recently with the discovery of new chemicals and synthetic products, the develop-

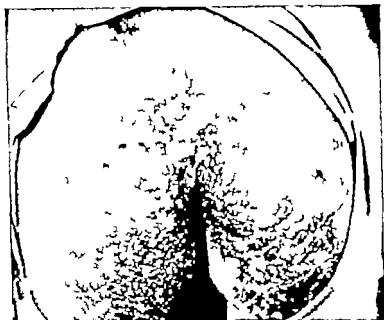


FIG. 29 Oil acne

ment of the patch test and interest on the part of the insurance companies. About 60 per cent of all industrial diseases are cutaneous disorders. Of these 10 per cent are cases of contact dermatitis. Over 1 per cent of all industrial workers are subject to occupational dermatoses.*

An occupational dermatosis is a pathologic condition of the skin for which occupational exposure can be shown to be a major causal or contributing factor.

The skin may be exposed to a (1) primary skin irritant, a substance which in a given concentration vehicle or manner of application produces a definite dermatitis in a person not previously sensitized to that substance or (2) the irritant may be a sensitizing substance which produces a skin reaction only in persons who are hypersensitive to that product.

*The standard reference book in this country is Schwartz, Louis, Tulipan, Louis, and Peck, S. M. *Occupational Diseases of the Skin*, ed. 2 Philadelphia, Lea & Febiger 1947.

General Etiology Predisposing causes include poor hygiene improper protection, chronic fatigue, excessive perspiration, heat, cold, friction photosensitization poor general health and nutrition a history of allergy hairy skin irritating soaps, cleansers and solvents and the presence of skin diseases (ichthyosis, dermatophytosis, seborrheic dermatitis, etc.)

Each industry and occupation presents its own special problems. The Negro is least susceptible, and his skin is least apt to react to intense heat, chemical irritants and photosensitizers.

Cutting oil dermatitis is often caused by dirty wipe rags, lack of protection of the worker outdated machines that splash oil on the worker and poor personal hygiene. Thorough cleansing of the exposed areas with a detergent containing wetting agents before leaving the plant may prevent oil folliculitis and acneform eruptions. Barrier creams are of no value in the prevention of this condition

Types of Eruptions and Their Cause Acute dermatitis usually is due to general or specific irritants chronic dermatitis (squamous or fissured) to fat solvents or detergents folliculitis to oils tar greases or chlorine and keratoses, to oils, coal-tar products, arsenic or anilines.



FIG. 294. Occupational hyperkeratosis of the soles in a patient with a dry skin. Long periods of standing on a cement floor was the cause.

Occupations Subject to Industrial Dermatitis

Housewives, cooks, dishwashers, soda dispensers, washerwomen. alkali dermatitis (soaps and powders) detergents and intertriginous dermatitis (moisture)

Bakers. salt, sugar flour baking-powder ingredients, diammonia

Masons and bricklayers. lime, cement, sand

Painters. paint, varnish, lacquers, turpentine, benzine

Tar and creosote workers. tar creosots

Dyers for fabric and leather dyes, fixatives, mordants

Road builders, farmers, gardeners plants, weeds, pollens and grasses

Photographers. hyposulfites, developers

Chromists and nickel platers. chromium and nickel salts

Nurses, dentists and physicians. Lysol, procaine, bichloride, picric acid

Laboratory workers. formalin, acid alcohol solutions

Manicurists. polish remover soaps, emulsions

Barbers. soaps, hair tonics

Filling-station attendants. greases, oils, detergents, gasoline

Lumbermen. creosote, sodium tetrachlor phenoxide

Machine shop workers. cutting oils, greases

Rayon industry. alkalis, viscose, chlorine

Chemical industry. acids, alkalis, salts of heavy metals, chlorine, etc.

Candy industry. sugar essential oils, flower nuts, chocolate, etc.

Fishing industry. water salt, trauma

Tannery workers. chromates, *B. anthracis*, sulfides

Dry cleaners. naphtha, gasoline, etc.

Rubber industry. chemical accelerators, etc.

Cannery workers. sugar, etc.

Insecticide industry. pyrethrum, arsenic, lead, mercury, etc.

Synthetic dye industry. sulfonic acid and intermediates

Physicians. mercury bichloride, procaine, iodine, formalin, rubber plaster of Paris, phenol, etc.

Nurses. streptomycin, thiotharsate of green soap, formalin, Lysol, iodine

Pathologists. formalin, alcohol, arsenic

Dentists. cocaine, procaine, soaps, mercury cresol, plaster of Paris, eugenol, wood alcohol, roentgen-ray

Veterinarians. iodine, mercurials, phenol

Druggists. essential oils, alkaloids, amics, thiotharsates, quinones, etc.

Diagnosis. A correct diagnosis is important because of compensation rights of the worker protection of the insurance carrier and proper management. A careful and complete history should be obtained from the patient and his foreman. If the patient did not have the eruption before he was employed. If the distribution is compatible with the exposure and if the suspected agent is compatible with the clinical type of the eruption, then you are on the right track. There is usually a history of improvement over the week-end or during vacations with recurrence following re-exposure. Patch tests are important and when positive offer suggestive evidence that the substance employed in the test is the sensitizing factor. If the eruption clears up when the patient

is removed from contact with the sensitizer no further proof is necessary

Differential Diagnosis. Contact dermatitis from irritants other than occupational, such as ointments, washing powders, hair dyes, etc., must be considered. *Dermatitis venenata* from plants and weeds may cause confusion. *Dermatomycosis* may spread or flare up as a result of irritation from local irritants. Nonoccupational dermatoses, including nummular eczema, disseminated neurodermatitis, pompholyx and toxic erythemas are often mistaken for eruptions of occupational origin.

In cases where difficulty arises, the following points must be kept in mind (1) a definite interval is usually present between the last exposure and the appearance of the eruption (2) the occupation is associated with a high incidence of dermatitis (3) the suspected agent has produced similar eruptions in other workers (4) the original site of the eruption corresponds to the site of maximum exposure to the suspected irritant (5) the eruption is aggravated by exposure to the suspected irritant and clears up when the patient avoids it for several days and (6) there is no other known cause for the presence of the eruption

Course. Some workers become "hardened" and after one or more attacks develop a permanent immunity to the irritant. These may be treated while continuing at work.

Others acquire only partial immunity and must be removed temporarily from contact with the irritant

A few develop a chronic dermatitis as a result of permanent sensitivity "broadening of the allergic base" as a possible result of a superimposed bacterial sensitization a cross-sensitization to chemically and biologically related products blotropism hyperirritability of the skin to local medication and interference with healing by persistent mechanical trauma (friction or scratching)

Chronic dermatitis also may persist in elderly workers and in those in poor health even though they have discontinued their occupations.

Prophylaxis. The use of gloves and tools when handling irritating substances is advisable. Protective (barrier) ointments have been formulated for specific industries but are more theoretically than practically useful. Instruction in the avoidance of careless exposure the use of mild or sulfonated soaps and detergents and early treatment of mild skin infections aid in reducing the incidence of industrial dermatoses.

No new substance should be introduced into an industrial process until its sensitizing or toxic properties have been studied thoroughly.

The incidence of occupational dermatoses may be combated by co-operation of the factory nurse the safety engineer the municipal and the state departments of occupational hygiene and in unusual cases the United States Public Health Service (Office of Dermatoses Investigation).

Treatment consists of (1) removal of patient from suspected environment, (2) soothing local therapy and (3) rest. Therapy is based on the general principles laid down for eczema.

Prognosis. In the average case if further contact with the irritant is avoided if preventive measures are instituted if cleanliness is insisted upon and if proper local treatment is prescribed, the eruption usually will disappear in a short time.

NUMMULAR ECZEMA

This is usually a form of indolent and recurring eczema which appears on the hands, the arms, the feet, the ankles and the buttocks as well-defined vesicular or papulovesicular quarter size or larger lesions. Involution is by central clearing. Recurrences are common.

Etiology. The condition appears to be a localized form of atopic eczema in patients with a psychosomatic background. However many of my cases occur in patients with a dry skin (xeroderma) and the lesions behave like a low-grade superficial infection.

Differential Diagnosis. The primary plaque of pityriasis rosea, fixed drug eruptions and dermatophytids of the eczematous type may cause difficulty.

Prognosis is poor for a permanent cure.

Treatment. The eczematous patches are hypersensitive to many drugs and in some cases to greasy ointments. Generalized autosensitization may occur from sensitizing medication. X-ray radiation (4 weekly doses of 75 r) have been effective in my practice. Wet dressings of 1 of 1 per cent silver nitrate solution or hydrocortisone lotion are useful in the papulovesicular types. Vioform, hydrocortone and tar ointments may be used in the dry types.

Infrequent bathing in the cold months and the use of superfatted soaps, and emollient oil applications after the bath, are useful preventive measures.

NURSING ASPECTS

The eczema group requires special care because of the hypersensitivity of the affected areas and the difficulty of controlling the pruritus which is often a prominent symptom. The patient should be completely disrobed in a comfortably warm room, and all involved regions should be noted. Dressings, if present, should be carefully removed according to orders from the physician. Since many patients with eczema as well as the relatives, are unusually alarmed and distressed the nurse should be calm, tactful and reassuring.

Atopic Eczema. Since itching is a primary symptom, the nurse should instruct the patient as to the application and the removal of all local preparations. Explicit instructions regarding baths, sedatives and the avoidance of allergens are important. Above all rest psychosomatic approaches and superficial psychotherapy are helpful. The nurse may gain from patients insights into personality disorders and etiologic factors which are denied the hurried physician. No injections drugs or local applications should be employed without the physician's orders. Dressings should not be loose enough to irritate the skin adhesive plaster on the uninvolved skin should be avoided and skin-colored lotions and ointments should be used whenever possible, to avoid embarrassment.

Infantile Eczema. These cases often present a therapeutic problem but proper nursing care often is rewarded with success. If special diets are ordered the nurse should observe weight loss or gain increase in the pruritus and effect on the stool frequency and character. Since the dirty-looking crude coal-tar ointment frequently is used in these cases, a supply of old clean sheets and linen strips rather than gauze is desirable. Sterile olive oil light mineral oil or Allertreme is used to remove the old salve. Any sign of irritation or secondary infection must be noted.

Restraints are necessary to reduce the trauma from scratching and rubbing which interferes with healing. Mittens are useless, and elbow cuffs and wrist restraints are unsatisfactory. Wrapping up the infant with arms and legs secured in a cotton blanket, often solves the problem. Sudden temperature changes should be avoided.

Infectious Eczematoid Dermatitis. The nurse should advise the physician of any spreading of the eruption or any evidence

of intolerance to local applications. Instructions regarding wet dressings should be typed and given to the patient. Separate tongue blades should be used to apply ointments in order to avoid contamination of the contents of the jar.

Varicose Dermatitis. The patient should be stripped to determine the state of nutrition, circulation and evidence of auto-sensitization. A stethoscope should be available, as well as tourniquets. If ulcers are present, the dressings should not be disturbed until examined by the physician. Ointments never should be applied directly to the dermatitis or ulcers but should be spread on muslin. Elastic bandages (2-inch or 3-inch) often are used after dressings are applied.

Contact Dermatitis. Under the direction of the physician, the nurse should obtain from the patient a list of possible contactants. Stock contactants or the original preparations or material often are used for testing, except in acute cases. The nurse should impress the patient with the danger of using home remedies, the importance of reporting any spread of the eruption from the prescribed treatment and the care of the patch test.

Drug Eruptions

STOMATITIS MEDICAMENTOSA
FIXED-DRUG ERUPTIONS

DRUGS IN COMMON USE WHICH
MAY PRODUCE ERUPTIONS
NURSING ASPECTS

DERMATITIS medicamentosa is an untoward cutaneous reaction resulting from the internal administration of a drug and apart from its therapeutic effect. This type of eruption is not to be confused with contact dermatitis, which is produced by the external application of a drug.

More than 100 drugs in daily use may cause eruptions with annual additions as new ones appear some useful some worthless, some put on the market without adequate clinical trial. They include not only the newer synthetic drugs but the common household and proprietary remedies. Therefore it is important for the physician to familiarize himself with the composition of all drugs prescribed and used by the patients.

Drug eruptions are as varied in their clinical manifestations as the drugs that cause them but a few by their appearance may give the experienced physician a clue as to the diagnosis. The same drug may produce a different type of eruption in different patients or in the same patient when prescribed at another time. On the other hand the same type of eruption may be produced by wholly unrelated drugs. The cutaneous reaction is capricious and unpredictable so that "typical drug eruptions are unusual."

In some cases the reaction is specific. In others it is a group reaction caused by members of the same chemical group while in others the reaction is heterophilic i.e. produced by members of different chemical groups.*

Drug eruptions are important for several reasons. They often are confused with the exanthemata of measles, scarlet fever, early

A more detailed discussion of the subject will be found in *Year Book of Dermatology* (1945) Part III pp 7 to 26

and late syphilis and other conditions. If the diagnosis is not made in the early stages of the eruption extensive involvement of the skin or an exfoliative dermatitis may develop. However most cases are mild and fleeting and only 0.5 per cent are severe enough to warrant hospitalization. There has been an occasional fatality from agranulosis (chloramphenicol) aplastic anemia (arsenic) or exfoliative dermatitis (penicillin)

Etiology The majority of the cases are caused by the development of a state of sensitization which in many instances is an antigen-antibody reaction. Overdosage resulting in a toxic state accumulative effect from long-continued dosage, impaired excretion as a result of kidney pathology and impaired detoxification from liver disturbances are also factors in the production of drug eruptions. Hypochondriacs epileptics and those who overindulge in self treatment are predisposed to dermatitis medicamentosa. The unnecessary use of penicillin and the sulfonamides provoke many cases of drug eruptions that could be avoided. The question "Have you ever broken out from any medicine?" always should be asked before writing out the prescription.

Biotropism the theory that some drugs may activate latent infections, may explain lichen-planuslike eruptions following arsenical medication and moniliasis and flare-ups of latent fungus infections during long-continued antibiotic therapy

Diagnosis. When a patient presents himself with an unusual eruption which does not resemble any of the orthodox eruptions the physician should become drug conscious. A cross-examination may or may not reveal the drug or drugs which are causing the eruption. It is not a good procedure to inquire of the patient

"Are you taking any drugs?" since he may erroneously interpret "drugs" as meaning narcotics and answer in the negative. More specific answers can be obtained by asking the patient what medication is used for headache constipation menstrual pains and other common conditions. The following points are important in the diagnosis of drug rashes

1. **HISTORY OF MEDICATION** with drugs self prescribed or obtained from a physician or a drugstore. Insistent questioning may be necessary and sometimes one cannot rely on the patient's denials.

2. **UNORTHODOX OR ATYPICAL ERUPTIONS.**

3. **TIME OF APPEARANCE.** Sudden appearance in hypersensit-

tive cases gradual development in accumulative types. Penicillin and iodides may provoke eruptions 7 to 14 days after they are discontinued (delayed reaction)

4 **THERAPEUTIC TEST** Fading of eruption when the suspected drug is eliminated. Aggravation or recurrence when a test dose is administered, although in the case of penicillin, gold and the sulfa drugs, the risk is not fair to the patient.

5 **LABORATORY TESTS** The drug may be discovered in the urine. Spectroscopic examination of incinerated tissues and microchemical tests of tissues sometimes are used to detect heavy metals. Complete blood counts are important in every case since some drugs cause a serious depression of the bone-marrow function

6 **BIOPSY** is of value only for differential diagnosis, except in bromoderma and iododerma in which the tissue changes are pathognomonic

7 **TONGUE TEST** In cases of angioneurotic edema, a small amount of the suspected drug is placed on the tip of the tongue, and the previous site of reaction observed for a flare-up.

8 **MUCOUS MEMBRANE TEST** In cases of codeine and the sulfa group sensitivities, a small amount of the drug is placed in the mouth. After a period under one half hour the eruption should be increased in intensity.

9 **PASSIVE TRANSFER TEST** In cases of drug hypersensitivity the bullous fluid may be used for testing normal individuals.

10 **INTRADERMAL TESTS** are of no practical value and, in the case of penicillin may be misleading.

Clinical Signs. Drug eruptions may be generalized or confined to localized areas. When localized, the distribution is determined by regional hypersensitivity (shock site) which in some cases is the area affected by a previous dermatosis.

Drug eruptions may mimic well-known diseases. Those due to barbiturates often resemble measles. acute arsenical eruptions simulate scarlet fever. iodides and bromides produce acne-form eruptions. quinine commonly causes urticaria, while pruritus often is produced by opiates.

Pathology There is no characteristic microscopic picture the changes varying from urticarial to nonspecific changes with hemosiderin or melanin. Verrucous lesions caused by bromides or iodides are typical with pseudoepitheliomatous hypertrophy and abscesses filled with P.M.N. leukocytes.

General Symptoms. The hypersensitive cases are usually free of general symptoms. Frequently the toxic cases are associated with fever, nausea, vomiting, rhinorrhea, salivation or diarrhea. Fever, restlessness, a fall in hemoglobin and leukocytes and a rapid pulse may occur in eruptions due to the sulfonamide group. Itching is usually absent, but pain is a constant symptom in bromide and iodide gummata.

General treatment consists of discontinuing the drug assuming it is known. If the patient is taking more than one drug and possibly an injection or two, all drugs should be avoided temporarily until the causative one is discovered. After the eruption clears, each of the drugs should be administered at 5-day intervals to determine which is the harmful one. In some cases the suspected drug may be necessary to save the patient's life but often a satisfactory substitute can be prescribed.

Measures to hasten elimination and suitable local therapy are necessary except in the mildest of cases. ACTH and cortisone may be effective in the acute hypersensitive types, if there are no contraindications (abnormal behavior, diabetes, hypertension or tuberculosis).

STOMATITIS MEDICAMENTOSA

Drugs may produce eruptions in the mouth which may mimic any oral disease. In most cases there are associated skin lesions but this is not always true.

The following is a list of drugs which may cause oral eruptions: Antipyrine-phenacetin group, arsenicals, barbital group, bismuth, halogens, mercury, phenolphthalein, sulfonamides, antibiotics, quinine and the salicylic-acid group.

FIXED-DRUG ERUPTIONS

Fixed-drug eruptions are those which reappear at the site of a previous eruption when the drug is readministered, even though small doses are given. Permanent vascular and pigmentary changes in the affected sites are responsible for this type of reaction. Phenolphthalein, arsenicals, antipyrine, phenytoin sodium, antibiotics, bromsulphalein, amidopyrine, sulfonamides, cinchophen, iodides and the barbiturates are the usual causes of eruptions of the fixed type.

Treatment. Detection and elimination of the drug and a mild soothing ointment usually results in clearing of the lesion within a few weeks.

DRUGS IN COMMON USE WHICH MAY PRODUCE ERUPTIONS

Acetanilid may cause extensive morbilliform, maculopapular or erythema multiformelike eruptions. The drug is present in Bromo-Seltzer and many "pain killers" sold in drugstores.

Acetarsons (See Arsenic)

Aconite produces an erythema or a macular eruption.

ACTH may produce anaphylactoid reactions in susceptible patients. Also urticaria, facial edema Cushing's syndrome water retention and psychoses.

Allonal (See Barbiturates)

Amidopyrine (See Pyramidon)

Aminopterin may cause diffuse alopecia.

Aminosalicylic acid (PAS) may produce general pruritus, scarlatiniform erythema, drug fever jaundice and gastrointestinal disturbances.

Antibiotics (See individual drugs)

Antihistaminics. Toxic erythema, fixed, pityriasis-rosealike, purpuric urticarial pruritic papular eruptions may occur. These drugs also may cause a flare-up of eczematoid eruptions.

Antimony drugs may cause toxic erythemas and exfoliative dermatitis. BAL is the drug of choice in treating these cases.

Antipyrine is a common ingredient of headache and anodyne preparations. Erythematous, bullous, urticarial and purpuric eruptions, fixed pigmented lesions and erosive balanitis may occur. The eruption may follow the first dose of the drug or develop after several doses have been taken.

Antitoxins (See Serum rashes)

Apresoline may cause toxic erythemas and erythema-multiformelike eruptions.

Arsenic in inorganic form formerly was prescribed frequently in psoriasis dermatitis herpetiformis and chronic eczema as Fowler's solution. Its danger lies in the fact that (1) certain individuals have an idiosyncrasy to this form of arsenic and (2) it usually is used over long periods of time often without supervision.

When the drug has been taken for a variable period the following disturbances may occur in hypersensitive individuals.

1. Epidermal hyperplasia, including arsenical hyperkeratosis of the palms and the soles, generalized pigmentation, multiple superficial epitheliomatosis and squamous-cell or basal-cell carcinomas.

2 Allergic reactions, consisting of urticarial, scarlatiniform, pityriasis-rosenlike, lichenoid and fixed erythematous eruptions.

3 Disturbances of the sympathetic nervous system resulting in herpes zoster and alopecia areata.

4 Blood dyscrasias, including purpura, acute hemolytic anemia and agranulocytosis.

5 Hepatitis.

Multiple superficial epitheliomas of the arsenical type occur as psoriasisform crusted indurated lesions with pearly borders. Histologically vacuolization of the cells and acanthosis are present. The appearance of this type of lesion has no connection with the amount of arsenic ingested or the duration of the arsenical therapy.

Arsenical keratoses frequently begin on the edges of the palms as a pink discoloration. While the palms, the soles and the dorsum of the ankles are the most common sites, keratoses may appear also on the fingers, the toes, the dorsum of the hands and the knees. The hyperkeratotic lesions may be scaly and

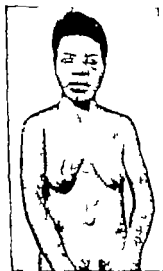


FIG. 30 (Left) Dermatitis exfoliativa from neocrophene. (Dr. Leon Goldman's case)

FIG. 31 (Right) Drug rash from phenobarbital sensitization

diffuse or they may consist of individual nodular or warty growths.

Cancer may develop in the keratotic lesions or in apparently normal skin if the carcinogenic properties of the arsenic have been awakened. Long latent periods have been reported as long as 40 years, from the time of ingestion of the arsenic to the appearance of the carcinoma. The predominating type is the squamous-cell cancer.

Arsenical melanosis is described under Pigmentations (See p 454)

TREATMENT Little can be done for the chronic types of inorganic arsenical eruptions. BAL and sodium thiosulfate are of doubtful value. Keratoses should be removed by excision or electrodesiccation. The melanoderma cannot be influenced by treatment but vitamin C medication and the avoidance of light seem to be of some benefit. If malignancy occurs, the lesions should be excised. The use of x rays or radium is not advised.

ORGANIC ARSENIC is present in arspenamine neoarsphenamine sulfarsphenamine acetarsone phenarsine hydrochloride, Maparsen Bismarsen Aldarsone and Tryparsamide. These drugs are now rarely used in this country.

ETIOLOGY The type of eruption produced by the organic arsenicals depends on the degree of cutaneous susceptibility and the local condition of the skin which may have been previously sensitized by an eczematous eruption, a fungus infection or seborrheic dermatitis.

The allergic or nonscaly type does not often follow the first injection but usually occurs after the second the third or the fourth injection. Sensitization may follow an accidental paravenous or intradermal injection or an intravenous medication following intramuscular injections of an arsenical.

The toxic or scaly types are caused by (1) large single doses, (2) too short intervals between treatments (3) arsenical retention and (4) poor excretion due to bad liver kidneys or heart.

CLINICAL TYPES The type of resulting cutaneous reaction is inherent in the patient's constitution.

The nonscaling types include the following varieties (1) urticarial (2) exanthematic—morbilliform scarlatiniform pityriasis rosealike ninth-day erythema poxialike, lichenoid and rose-troid eruptions (3) purpuric (4) fixed.

The *scaly type* is essentially an exfoliative dermatitis the various clinical types depend on the degree of involvement.

DIAGNOSIS is based on the history of arsenical medication which is usually sufficient evidence. In some cases where other drugs have been used or occupational and other factors enter the picture laboratory data may be necessary to establish the diagnosis. Intradermal and patch tests are of limited value and require interpretation by the expert. In medicolegal cases, qualitative tests on the hair the skin and the urine usually are made. Osborne's method of microchemical visualization is not always successful.

Danger signals of impending dermatitis include malaise fever chills, generalized pruritus, unexplained erythema and desquamation of the palms and the soles.

PROGNOSIS. Toxic hepatitis, hemorrhagic encephalitis, uremia and bronchopneumonia are serious complications. The mortality rate among a group of 139 cases of arsenical dermatitis (Co-operative Clinic Group) was 2.9 per cent.

TREATMENT The patient should be hospitalized and kept comfortable by standard nursing procedures. BAL (British-Anti-Lewisite) is the drug of choice but must be used early. The dose is 0.025 cc. of the ampul solution per Kg body wt. repeated at 4-hour intervals for a total of from 4 to 6 injections on each of the first 2 days. Thereafter 2 injections daily until recovery are sufficient. Nausea and vomiting, as well as painful abscesses, may occur.

The liver should be protected and dehydration prevented by daily intravenous injections of glucose choline high protein diet and hydrocortisone. If severe anemia is present blood transfusions are advisable.

A *salt free diet* is indicated to reduce the moisture content of the skin and to prevent renal irritation. If stomatitis is present, the diet should be liquid but nourishing. A high-vitamin high-caloric diet is necessary during convalescence. Large doses of ascorbic acid are useful.

Local treatment consists of calamine liniment or equal parts of olive oil and lime water in the dry cases starch-powder baths or mild astringent lotions in the moist cases. Denuded areas should be painted with 0.5 per cent aqueous solution of brilliant green.



FIG 32 Bromoderma

FOLLOW UP After the patient recovers, the question of further antisyphilitic treatment must be considered. At least 8 weeks should elapse before resuming treatment in order that the patient may regain his weight and build up his resistance. Treatments should be resumed with penicillin because of its relative safety and effectiveness.

Aspirin (See Salicyl compounds)

Asterol which is used externally in ointment and tincture may cause toxic erythemas, convulsions and neurotoxic reactions in children

Atabrine (See Quinacrine)

Atophan occasionally produces a scarlatiniform erythema.

Aureomycin may produce angioneurotic edema stomatitis, glossitis, vulvitis and anal pruritus.

BAL may cause a feeling of constriction in the throat and a burning of the tongue

Barbiturates are prescribed extensively as phenobarbital, Luminal Veronal Barbitone Seconal Amytal Nembutal and pentobarbital sodium. They may aggravate any pre-existing dermatosis. They produce scarlatiniform or morbilliform eruptions

which may leave a residual pigmentation. Bullous eruptions with pyrexia, erythema multiforme and even exfoliative dermatitis may result. Fresh attacks occur following each successive dose. Substitute rauwolfia preparations in sensitive patients.

Belladonna and atropine rashes are common in children. Bright red scarlatiniform eruptions may involve the face and the neck, persisting for a few days after the drug is withdrawn.

Bismuth. Bismuth pigmentation of the oral mucosa and stomatitis were common during the prepenicillin era. Pruritus, urticaria, herpes zoster lichenoid and erythema-multiforme eruptions have been reported.

TREATMENT. BAL is the drug of choice and is used according to the technic described under Arsenical Eruptions.

Bromides produce eruptions similar to those caused by iodides. The potassium salt is more likely to produce rashes than the other salts. Children are apt to suffer severe effects. When prescribing bromides or iodides physicians should not overlook the fact that infants may absorb the drug from the mother's milk. Bromo-Seltzer, Miles Nervine Tonic and numerous proprietary "nerve tonics" contain the drug.

Epileptics who require bromide sedation over long periods are subject to bromide rashes. In extensive cases of bromoderma, albumin and casts may appear in the urine as a result of renal irritation.

VARIETIES. Bromoderma consists of four important types of eruptions: the acneform, the furunculoid, the granulomatous or vegetating, and the bullous or pemphigoid variety.

DIFFERENTIAL DIAGNOSIS. The acneform type is similar to acne, but no comedones are present. Bromide granulomatous tumors which resemble gummata usually appear on the legs, the buttocks and occasionally on the face. They are characterized by tenderness, slow development, persistence and gradual evolution. They must be differentiated from syphilitic gummata, rupoid psoriasis and blastomycosis. The bullous variety may resemble pemphigus, but the inflammatory areola, the absence of systemic involvement and the presence of bromides in the urine and the blood are confirmatory. Toxic symptoms of overdosage include mental confusion, stupor, delusions, headache and hallucinations.

TREATMENT. (See under Iodides)

Butaxolidin, an antiharthritic drug may cause urticaria and toxic erythemas.

Choral hydrate may produce eruptions of the erythema type, including urticaria, petechial hemorrhages, scarlatiniform and morbilliform lesions.

Chloroquine (Aralen) may cause inability to accommodate.

Cinchophen may cause urticarial bullous or erythematous eruptions.

Codeine (See Morphine)

Cortisone (See ACTH)



FIG. 34 Iodide eruption (vegetating type)

Diazone Vacular papular and pemphigoid eruptions have been reported.

Digitalis rarely produces drug eruptions although it is frequently prescribed in patients with poor circulation which affects the oxidation and the excretion of the drug. Erythematous, papular and scarlatiniform eruptions may occur



FIG. 34 (Left) Balloon iodide eruption.

FIG. 35 (Right) Iododerma. These vegetating lesions closely resemble those of late syphilis. (Dr. Leon Goldman's case)

Dilantin sodium which is widely used in epilepsy may produce a hypertrophic gingivitis, toxic erythema purpura and rarely exfoliative dermatitis.

Germanin has been used in pemphigus and dermatitis herpetiformis. Generalized papular eruptions have been described.

Gold in the form of the sodium thiosulfate salt Sanocrysin Solganal, Myochrysin or Krysolgan, was popular until the advent of steroid therapy in lupus erythematosus, arthritis and ocular tuberculosis.

Contraindications are acute pulmonary tuberculosis, liver and kidney disease. Pruritus, morbilliform, scarlatiniform and lichenoid eruptions are not infrequent in sensitized individuals. Any of these types may develop into an exfoliative dermatitis with fever arthritis, albuminuria and stomatitis. Pigmentation usually follows the exfoliative type. Fewer reactions result if an initial

Butazolidin, an antiarthritic drug may cause urticaria and toxic erythemas.

Choral hydrate may produce eruptions of the erythema type including urticaria petechial hemorrhages, scarlatiniform and morbilliform lesions.

Chloroquine (Aralen) may cause inability to accommodate.

Cinchophen may cause urticarial bullous or erythematous eruptions.

Codeine (See Morphine)

Cortisone (See ACTH)



FIG. 13 Iodide eruption (vegetating type)

Diazone Macular papular and pemphigoid eruptions have been reported.

Digitalis rarely produces drug eruptions, although it is frequently prescribed in patients with poor circulation which affects the oxidation and the excretion of the drug. Erythematous, papular and scarlatiniform eruptions may occur.

doses of ascorbic acid mild astringent lotions and antihistaminic drugs are also useful. In the presence of agranulocytosis, penicillin by injection must be used as soon as the diagnosis is made.

Insulin may produce localized areas of lipodystrophy at the site of injection, urticarial and morbilliform eruptions.

Iodides. The iodide salts, especially potassium iodide are ingredients of many proprietary "tonics," iodized salt, "rheumatism" and arthritis mixtures. Iodized oil is used in the diagnosis of chest conditions. They are frequent causes of drug eruptions.

The acneform type may mimic acne vulgaris, but the lesions are more inflammatory, larger and spread downward from the usual sites of acne to the thighs and the legs. Iodides and bromides also may produce lesions of erythema nodosum in susceptible individuals. Less frequent but more important because of diagnostic difficulties are the iodide gummata, which are usually multiple and resemble syphilis, carcinoma or tuberculosis. Coryza, gastro-intestinal disorders, toxemia and cachexia may occur in the extensive cases of iododerma.

DIAGNOSIS is made by history, distribution, examination of the urine and therapeutic test. After elimination of the drug, the acneform lesions clear up in a few weeks, but the gummatus types are persistent and involute very slowly.

TREATMENT OF IODERMA AND BROMODERMA. As soon as the diagnosis is made, the patient should receive an intravenous injection of 100 cc. of normal physiologic saline solution. This should be continued daily until the lesions involute and the drug has been eliminated from the tissues. After six or more injections, ammonium or sodium-chloride tablets (5 to 10 Gm. daily) should be prescribed for a week or longer provided there is no contra-indication (severe cardiorenal disease or hypertension). Fluids should be forced to hasten elimination. If medication must be continued, other drugs having similar therapeutic properties should be prescribed. Lotio alba is useful in the acneform types of eruption.

Luminal (See Barbiturates)

Isonicotinic acid may produce urticaria and Herxheimerlike phenomena in susceptible patients.

Mapharsen may occasionally produce jaundice, pruritus, erythema, fixed eruptions and exfoliative dermatitis.

Mercupurin may cause stomatitis, urticaria, fever and anaphylactic shock.

Mercury rarely causes eruptions when ingested or injected, but dermatitis and pigmentation are common from the use of solutions and ointments containing the drug.

Methyl and propylthiouracil may cause erythema multiformelike eruptions erythema nodosum urticaria, fever and alopecia.

Morphine and Opium. The common reaction to these drugs is extreme itching. Therefore it is inadvisable to use morphine or its derivatives in the treatment of pruritus. Urticarial and scarlatiniform eruptions are rare.

Penicillin reactions may be immediate or delayed. The immediate reactions, which occur within 24 hours, consists of erythema, urticaria vesicular "id" lesions on the hands and the fingers angioneurotic edema, purpura, flare-up of pre-existing eruptions generalized eczematoid eruptions and Herxheimer reactions in syphilis. Edema of the face also may appear.

Delayed reactions (about 6 per cent) consist of generalized urticaria angioneurotic edema myalgias edema about the small



FIG. 37 (Left) Pigmented macular phenolphthalein eruption (Dr. C. D. King case)

FIG. 38 (Right) Sulfapyridine eruption

joints and serum sickness. These reactions may occur from 7 to 14 days after the start of treatment.*

Antihistaminic drugs and intravenous calcium (Calglucon—Sandoz) therapy reduce the severity of the attacks and control the pruritus. In severe cases, ACTH hydrocortisone or prednisolone therapy should be considered.

PREVENTION OF PENICILLIN REACTIONS. Skin testing for the detection of hypersensitivity has not been uniformly successful. It is not possible to prevent reactions entirely but the following are important precautions: shun its use in asthmatics; avoid indiscriminate use; give antihistaminics before reactions occur in those with allergic histories; previous sensitizations or presence of fungus infections; desensitize with small doses when possible; use of hypo-allergenic penicillin (Duracillin [Lilly] Penicillin PBZ [Ciba] Cer-o-cillin [Upjohn] and Compensamine [Commercial Solvents]). The author requests his penicillin-sensitive patients to wear a metal tag containing the inscription "I'm allergic to penicillin."

Phenacatin medication may result in urticarial and erythematous eruptions as well as localized edema. Anacin and empirin compound which are widely used anodynes, contain the drug.

Phenobarbital may cause pruritus morbilliform, urticarial and purpuric eruptions, oral ulceration exfoliative dermatitis and jaundice.

Phenolphthalein is widely prescribed as a mild laxative. Several hours or days after the drug is ingested, itching and burning develop at the site of the eruption. At first the lesion is a bright red macule that changes from day to day to a dark red, purple brownish-red or dark brown color. Usually these polychromatic macules are well defined and range in size from a dime to a dinner plate. In most cases the trunk, the arms and the wrists are affected but the face, the neck and the genitalia also may share in the involvement. Repeated ingestion of the drug usually produces a flare-up and a violaceous tinge to the lesion.

Eruptions of the fixed or persistent type in which flare-ups occur after each dose of the drug, generalized pigmentation and single or multiple erosions in the mouth and on the genitalia are not uncommon. The drug also may cause urticaria in children and adults with the production of large wheals which often leave

*For a broader review of the subject, consult Lane S. L. Reactions from antibiotics and the sulfas, *Ann. Allergy* 11-615 1951.

residual pigmentation. In recent years, reactions to the drug are seen rarely.

DIAGNOSIS If the lesion under observation flares up with increased redness and pruritus after a test dose of one grain of the drug daily for two days, the evidence is suggestive of a phenol phthalein eruption.

Phenytol sodium may produce scarlatiniform, morbilliform, bullous purpuric and fixed eruptions.

Pyramidon produces localized or generalized erythematous or urticarial eruptions. Purpura, pruritus and localized edema of the eyelids and the lips have also been noted.

Quinacrine hydrochloride (Atabrine) (see p. 120) may produce lichen-planuslike lesions, eczematoid dermatitis, exfoliative dermatitis and the typical yellowish discoloration which appears after the second week.

Quinidine. Thrombocytopenic purpura and lichen-planuslike eruptions have been reported.

Quinine. This drug is extensively prescribed in malaria, colds and febrile conditions, as *Coco-Quinine*, *Bromo Quinine*, *Hill's Cascara Quinine*, *febrile Chloroquin* and many other preparations. It has photosensitizing properties which are probably a factor in the production of the cutaneous reactions. The common eruption is a widespread urticaria. Erythematous papular vascular and bullous eruptions are more unusual and may involve the face, the trunk and the palms. In some cases, purpuric lesions may appear on the oral mucous membranes.

Rauwolfia (*Serpasil*, *Raudixin*, *Reserpine*) may cause purpura after prolonged use.

Salicyl Compounds. These include aspirin, sodium salicylate and salicylic acid. Angioneurotic edema, urticaria, purpura, salivation and vasomotor rhinitis are not uncommon from salicylates.

Santonin may produce erythematous, urticarial or morbilliform eruptions.

Sedormid, a carbamide sedative, may cause thrombocytopenic purpura.

Serum rashes occur from sensitization to foreign proteins in *Hapamine*, horse serum, antitetanic, antidiphtheritic and antistreptococcic serum. Eruptions may result from one injection of the product or repeated doses. Simple serum reactions consist of localized edema, urticaria, erythema multiforme or purpura which may occur from 24 to 48 hours after injection. Immediate relief

is obtained with adrenalin or intravenous injection of 50 cc. of a 50 per cent dextrose solution. The antihistamine drugs are useful in controlling the condition and relieving the pruritus. ACTH or cortisone may relieve symptoms in severe cases.

Silver (See under Pigmentations, p 455)

Stilbamidine may cause fifth-nerve damage in susceptible patients.

Streptomycin, when used for extended periods, may produce reactions (7 per cent) including urticaria, erythroderma, bullous eruptions and stomatitis.

Sulfonamides. About 6 per cent of all patients treated with these drugs show some evidence of cutaneous intolerance. Urticaria is not uncommon and appears to be activated by exposure to sunlight. Next in frequency are the toxic erythemas, including measleslike and scarlet feverlike eruptions, maculopapular lesions, erythema multiforme diffuse erythemas and purpura. The involvement may be limited to a generalized pruritus edema of the eyelids or erosions in the mouth.

Systemic symptoms consist of fever, dizziness, prostration pain in the chest, headaches and sneezing.

Blood dyscrasias include acute hemolytic anemia, methemoglobinemia and agranulocytosis. When the polymorphonuclear leukocytes fall below 50 per cent the drug should be discontinued.

Sulfocyanates. The potassium salt was formerly used in cases of hypertension. Urticarial morbilliform papular and exfoliative dermatitis eruptions may occur.

Testosterone. Overdosage in the female may result in acne-form eruptions, hypertrichosis and other signs of virilism.

Tridione, an anticonvulsant, has been reported to cause herpetic lesions and superficial ulcers in the mouth and on the genitals.

Veronal (See Barbiturates)

Vitamin A, when its use is prolonged in susceptible patients, may cause alopecia, pigmentation over areas exposed to light, general pruritus, joint pains and tenderness over the long bones.

Prevention of Drug Eruptions. Often the incidence of this complication can be avoided by (1) obtaining a history of previous sensitization, (2) avoiding potent drugs unless absolutely necessary (3) substituting less toxic drugs when possible (4) prescribing an antihistamine when giving gold, penicillin etc., (5) acquainting the patient with the early signs of drug intoler-

ance and (6) discontinuing the drug at the first sign of hypersensitivity

When the physician is aware of the fact that the patient is sensitive to a certain drug a warning note in large red letters on the history card will prevent future embarrassment.

NURSING ASPECTS

While most drug eruptions are mild and evanescent some may be *disabling and even fatal*. The nurse should be familiar with reactions from penicillin and barbiturates. The hospital nurse should have experience with intravenous techniques and BAL therapy employed in drug eruptions caused by heavy metals. Any unusual eruption occurring in a patient who is receiving oral therapy or injection therapy should be reported at once to the physician.

Psoriasis

Definition. Psoriasis (the name is derived from the Greek word *psora* the itch) belongs to the group of chronic erythematous squamous eruptions. It is a relapsing disease of unknown etiology consisting of sharply defined dry patches of erythema covered with silvery scales and having a capricious course. The disease is controllable but not curable. It involves the skin and the nails, but it does not affect the hairs or the mucous membranes. There are usually three stages (1) evolution or progression (2) the quiescent stage and (3) retrogression or improvement.

Clinical Description. The eruption consists of scaly plaques of various sizes limited or generalized in distribution.

The primary lesion is a pinhead-sized, dry round or oval, sharply defined and slightly elevated papule covered with a silvery or frosty scale.

THE PLAQUES are formed by an increase in the size of the papule or by a coalescence of older lesions and usually occur in irregular shapes. They are always dry flat infiltrated and covered with loosely adherent glistening scales.

THE SCALES are dry, thin slightly translucent frosty silvery or asbestoselike and lustrous white in color. Analysis of the scales

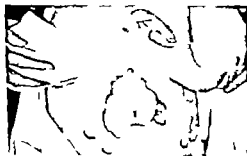


FIG. 39 Annular psoriasis.

has shown them to have a high cholesterol content. When the scales are removed with the nail or with a blunt instrument, they leave minute bleeding points (Auspitz sign) corresponding to the tops of the papillae of the corium. However this sign also is found in other scaling conditions.

The amount of scaling in a given case depends on the following facts. Scaling is decreased in acute eruptions and as a result of moisture sweat frequent bathing medication and friction. Scaling is increased in the presence of a dry skin, poor hygiene, in chronic cases and in old age.

ITCHING is neither a prominent nor important symptom. The acute types are often pruritic because of the inflammatory element.

Various descriptive terms have been applied to the clinical types depending on the character of the predominating lesions. Punctate psoriasis is composed of pinhead sized lesions. The discoid type consists of coin shaped plaques. The guttate type

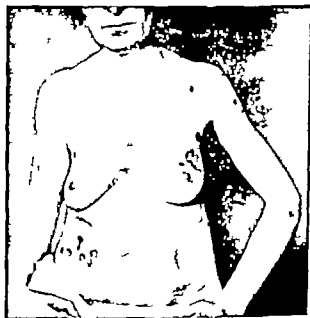


FIG 40 Chronic psoriasis. The lesions usually vary in size and shape.

is of drop size the geographic type has a wavy or spreading edge. The circinate type predominates in annular or ring forms the rupoid type consists of thickened and crusted lesions. Linear psoriasis (nevus or birthmark type) is characterized by bandlike lesions, with a nevus distribution, which wax and wane unlike a nevus. However no nevus cells are present.

Localization. Psoriasis is one of the few diseases that is characterized by a selective localization, which makes the diagnosis relatively easy. The eruption is usually bilateral. It may be generalized but it is never universal except when complicated by an exfoliative dermatitis. An important feature of the disease is its tendency to involve the extensor surfaces. It prefers the elbows, the knees, the anterior aspect of the legs, the scalp and the sacral regions.

When the scalp is involved the patches are edged with erythema and are covered with thick, white mortarlike scales which are impossible to remove with soap and water or by scraping. The lesions affect the hairy parts but avoid the bald areas. Psoriasis never causes alopecia.

The genitals, the intergluteal cleft, and the inframammary regions also may be involved. When psoriasis appears in these locations, friction and moisture alter the characteristic features of the disease. There is a minimum of scaling. The palmar and the plantar types are characterized by hyperkeratosis fissures and resistance to therapy. When the face is involved (generalized cases) the patches usually are indistinct small and ill-defined, with very little scaling present.

The nails are involved in about 25 per cent of all cases. Nails may present three types of lesions (1) pitting as a result of local punctate injury to the germinal epithelium of the nail (2) yellowish or porcelain-colored opacities, and (3) chalky subungual hyperkeratosis. The fingernails are involved twice as frequently as the toenails. Pitting is not pathognomonic. It also occurs in rheumatism, tuberculosis and chronic fevers. Usually treatment is ineffective.

Unusual Types of Psoriasis. **SEBORRHEIC TYPE.** Psoriasis may stimulate seborrheic dermatitis by involving the scalp the face and the flexural surfaces to a greater or a lesser extent. The features of seborrheic dermatitis may predominate at one time those of psoriasis, at another. Biopsy is not helpful in differential diagnosis. This type is resistant to treatment.



FIG. 41 Chronic psoriasis. This is a typical site. Note the sharp borders, the infiltration and the silvery scaling.

PUSTULAR TYPE. This is a rare form of pustulation and crusting that may appear in old patches of psoriasis. Usually generalized psoriatic lesions are found elsewhere but their presence is not required to fulfill the diagnosis, although the pus must be sterile. Pustules in this type are caused by the formation of exaggerated lakes of microscopic Monro abscesses in the epidermis with the formation of macroscopic abscesses that dry up and form crusts. The characteristic psoriatic pathology is absent during the pustular stage. This type is resistant to therapy.

ARTHROPATHIC TYPE. About 5 per cent of all cases of psoriasis have associated joint pathology that Madden and Karen believe is coincidental. They concluded that there is no such thing as a psoriatic joint based on roentgenogram evidence alone. In the arthritic type the joint involvement usually consists of the characteristic rheumatoid arthritis with pronounced bone destruction in some cases (O'Leary). It is common for the psoriatic attacks to coincide with each recurrent flare-up of the joint disease. The terminal interphalangeal joints are the first ones to be affected.

ABERRANT (ATYPICAL) TYPE. In some types of psoriasis, lesions may be present which resemble those of localized neurodermatitis, eczema, seborrheic dermatitis, palmar epidermophytosis, tinea cruris or exfoliative dermatitis. These psoriatic lesions are dependent on general factors, seborrheic soil and various

local conditions which may modify the characteristics of the disease.

PALMAR TYPE. Often mistaken for "ringworm" psoriatic lesions may involve the mid-palms the finger tips and the knuckles. Fissures are not unusual. In most cases an examination will reveal a small patch on the elbows, the knees or the sacral region which the patient forgot to mention. Response to therapy is poor.

INVETERATE TYPE. About 1 per cent of all cases of psoriasis belong in this group. The lesions are chronic, refractory to treatment and persistent. Patients with this type usually wander from doctor to doctor or disregard it entirely.

ACUTE TYPE. Acute psoriasis is difficult to diagnose in its early stages because of the marked inflammation and the absence of scaling. The lesions are brighter red less sharply defined less scaly and more pruritic than those of the chronic type. The acute form generally terminates after weeks or months as the chronic variety.

NAIL TYPE. In rare cases the disease may be limited to the nails with cloudy yellow patches and heaped-up hyperkeratoses under the free edges of the nails.

Etiology. Since the cause of psoriasis is unknown the physician may satisfy the patient's curiosity by explaining that the disease is a "form of birthmark."

The following theories of causation have been advanced (1) cerebral corticoseomatic depression as a result of various trigger mechanisms causing a decreased cellular oxidation (Charpy) (2) metabolic (disturbance of lipid metabolism) (3) endocrine (4) adrenocortical hormone disturbance because of improvement during pregnancy and beneficial effects of ACTH and cortisone in psoriatic arthropathy (5) light starvation and (6) lack of adaptation to stress. The fact that in most cases the disease is first noted in the scalp suggests a close relationship to seborrheic dermatitis which may stimulate acute psoriasis.

The consensus at the present time is that psoriasis is a peculiar cutaneous reaction to various factors with the production of a parakeratosis on a favorable soil.*

The incidence varies from 4 to 6 per cent of all skin diseases. There appears to be a familial or hereditary incidence in about

*The current theories and experimental work has been reviewed by Gane, O. Some observations on pathogenesis of psoriasis, *A.M.A. Arch. Derm.* 85: 598-611, 1952.

2 per cent of my cases a predisposition rather than a genetic factor is involved. The average age of onset is 20 years the disease is uncommon in persons under 5 years and over 60 years of age. Psoriasis is very rare in full blooded Negroes and other dark races. There is a tendency for the disease to develop after minor traumatism, such as friction irritation or injury (*Koebner isomorphic phenomenon*) Some cases are precipitated or aggravated by exposure to the sun, acute infections psychic trauma and physical injury

Pathology There is no absolute pathognomonic histopathologic criteria in psoriasis. Neither is there general agreement as to whether the disease begins in the epidermis or in the corium.

The epidermis shows (1) a hyperkeratosis and (2) a uniform parakeratosis, (3) absence of the granular layer (4) a regular acanthosis with slight intercellular edema of the prickle cells, (5) a marked downgrowth of the interpapillary projections and (6) groups of migratory leukocytes in the horny layer (Monro pseudo-abscesses) The corium is the site of (1) vascular dilatation (2) elongation of the papillae and (3) a perivascular infiltration consisting of lymphocytes, polymorphonuclear cells and fibroblasts.

Chronic seborrheic dermatitis may resemble chronic psoriasis, but the presence of a greater amount of edema, less parakeratosis, the absence of Monro abscesses and the presence of areas of spongiosis are important points in differential diagnosis. In localized neurodermatitis, the granular layer is present and the acanthosis is irregular

Lichen planus when it is limited to the legs especially is apt to cause confusion with psoriasis. Usually the eruption behaves badly if tar or anthralin ointments are used, a fact that should warn the physician of a possible error in diagnosis. A biopsy of a typical lesion should establish the diagnosis.

SEQUELAE. Eczematization may occur in certain individuals as a cutaneous reaction from (1) friction or moisture or (2) the local effect of therapy. This reaction consists of redness and weeping. Its presence is sufficient reason for changing the type of treatment. Lichenification may occur in chronic pruritic lesions and cause treatment resistance.

Exfoliative dermatitis or its milder form, *erythroderma psoriatum*, may appear independently or may follow the use of strong ointments, viz tar chrysarobin or ammoniated mercury

This is a peculiar cutaneous reaction characterized by generalized scaling and redness, disturbance of hair and nail-growth chilliness, anemia and sometimes albuminuria. The histology retains the features of psoriasis, so that differential diagnosis from lymphoblastoma is possible. This accident need not occur if patients are observed at frequent intervals and informed of the early signs of impending erythroderma (spreading erythema beyond the sites of the original psoriasis, itching and general irritability)

The development of skin cancer in psoriatic patches is rare. When it does occur it is usually of the squamous-cell variety. It may result from long-continued medication with arsenic or excessive x-ray radiation.

Diagnosis. Psoriasis usually can be diagnosed by observing the following salient points:

- 1 Involvement of extensor surfaces, elbows knees, scalp and lumbosacral areas.
- 2 Scaly dry red plaques with sharply defined borders.
- 3 Infiltrated lesions covered with white, silvery scales which leave bleeding points when flicked off.
- 4 A history of previous attacks.
- 5 Improvement during summer months, frequently but not always.
- 6 Absence of itching.
- 7 Frequent involvement of the scalp without loss of hair.
- 8 Pitting or discoloration of the nails in some cases.

Differential Diagnosis

	<i>P. arvensis</i>	<i>Seborrheic Dermatitis</i>	<i>Physicis Rases</i>
Scales	Dry and silvery	Greasy yellowish or crusty	Thin and flaky; scaly collar around margin of lesion
Bleeding points	Present	Absent	Absent
Location	Extensor surfaces	Flexural surfaces	Trunk
Surface	Dry	Greasy	Dry
Color	Erythematous	Orange yellow	Salmon-color
Induration	Moderate	Slight	None

Prognosis. The patient must be informed that psoriasis is controllable but not curable. It is impossible to determine how long an interval will elapse between attacks. About 20 per cent of the cases undergo complete remissions regardless of the type of treatment. The remission may last weeks, months or years, a greater period of freedom occurring if all lesions disappear following treatment. About 60 per cent of the cases show remissions during the summer months.

Walker and Percival are convinced that one of the reasons for the unfavorable prognosis is that often an unfavorable prognosis is given. We agree that a patient with psoriasis will not continue with the strenuous therapy necessary if the physician shows the least bit of scepticism and lack of enthusiasm.

Treatment. No patient should be treated for psoriasis unless he is willing to accept the treatment outlined for him nasty and messy and time-consuming though it may be. No pill diet or shot is available at this time that takes the place of the appropriate local medication. It is necessary that the general health of the patient be maintained. Inquiry should be made of any previous treatment the patient has received and of any reactions from that treatment.

Practically every drug chemical and endocrine product has been used with varying results. Any new type of treatment helps occasionally especially in first attacks, the rapidity of improvement depending on the enthusiasm of the physician.

Every accessible endocrine gland has been irradiated vitamins have been used in huge doses sex hormones and placental extracts have been advocated by European observers, but the mystery of psoriasis remain.

DIETETIC. There is no basis for the use of specific dietetic methods although acute cases in general do better on low-protein diets. A strict hospital regimen with suitable dietetic measures appears to affect the metabolism of psoriatics favorably and hastens the response to local measures. Alcohol should be forbidden since it may make some cases treatment-resistant.

INTERNAL MEDICATION is rarely necessary or useful. Alkalis the citrated carbonates and the barbiturates appear to be useful in the acute cases. Arsenic should not be used in the treatment of psoriasis for the following reasons: (1) it is effective in only a small percentage of all cases (2) because of the chronic nature of the disease there is danger of accumulative action with possible ar

enical melanosis or epitheliomatosis and (3) other measures are safer and more effective. In the arthritic types of psoriasis hydrocortisone and ACTH have produced good results. When these drugs are used the patients should be hospitalized and placed under close supervision. These hormones do not benefit the common types of psoriasis.

Other chemotherapeutic agents include colloidal sulfur in 5-cc. doses intravenously twice a week or 2 cc. intramuscularly and intravenous injections of sodium salicylate (20 per cent) which do not give encouraging results except in the acute arthritic types.

FOREIGN PROTEIN THERAPY The safest preparation and one often effective when used in conjunction with local therapy is whole blood (autobemotherapy). From 10 to 20 cc. of blood is injected twice a week in a course of from 10 to 20 doses. It is a preferred collateral treatment in children and in the aged.

Triple typhoid vaccine is a valuable procedure in those cases



FIG. 42 Chronic psoriasis. Not thick, white-dry mortarlike scales. Patient was chronic alcoholic.



FIG. 43. Psoriasis of the pubic area with involvement of the vulva. The crumbly character of the lesions suggests a fungus infection. Typical psoriasis lesions are present also on elbows, back, and knees.

that resist local measures. It is administered intravenously every other day in the following doses 50 100 200 400 500 million. This treatment is contraindicated in tuberculosis and pregnancy and in liver cardiac, kidney and hypertensive diseases. The patient should be hospitalized and kept in bed during the course of injections. The temperature usually reaches 102 to 104 F within an hour after the injection. Headache, backache and pains in the legs occur but disappear within 24 hours. No antipyretic drugs should be prescribed but codeine may be given to relieve the side-effects. A 3 per cent chrysarobin ointment should be used on the body and a 6 per cent ammoniated mercury ointment on the scalp during the treatment.

PHYTOTHERAPY The sun is the psoriatic's best friend (Stokes). General exposures with gradual pigmentation will often clear up resistant lesions and increase the intervals between attacks. For tunately is the psoriatic who can spend a few weeks in southern Florida or California or the Caribbean area in the winter.

Ultraviolet light exposures are beneficial in the average case during the winter months. If care is used in gradually increasing the dosage to tolerance and avoiding "burns." Often they are useful in conjunction with the coal tar treatment. This form of therapy should not be used in acute cases, which may become aggravated. Although this treatment fails in some cases and succeeds in others, the best results are obtained when the light is used daily. Unless there is subsequent tanning of the skin, the eruption is apt to persist. My experience with self treatment at home with the small portable ultraviolet lamps has not been satisfactory.

X ray irradiation never should be used in the treatment of psoriasis even though the dermatologist may have an urge to do so in the therapy of localized patches. Results are too unpredictable and the danger of late roentgen dermatitis always is a possibility. For persistent scalp and scrotal lesions, Grenz rays may be employed by the expert.

BALNEOTHERAPY In resistant cases, a course of baths at a thermal resort (e.g. Hot Springs, Ark.) may give the patient some relief at least to his morale.

LOCAL TREATMENT is most dependable. The patient should be warned regarding the possibility of an erythema from ultraviolet light therapy or a mild dermatitis from sensitization to one of the ingredients in the applications (tar sulfur ammoniated mercury salicylic acid, Anthralin). If sensitization occurs, therapy

should be changed and a bland ointment or soothing lotion applied for a few days. Close supervision is therefore necessary in all cases under active therapy. It is best to respect the wishes of the patient as regards the therapeutic program.

Acute Cases Burow's zinc emulsion or Nivea oil should be used in treating acute cases. Astringent applications such as calamine lotions or zinc oxide-starch mixtures also are useful.

<i>Liquor carbonis detergens</i>	5.0
Zinc oxide	16.0
Corn starch	16.0
Magma bentonite	40.0
Lime water q.s. ad	100.0

Bed rest, sedation, autohemotherapy and colloid baths are often necessary to obtain relief. Stimulating local therapy is contraindicated to avoid the possibility of spreading of the eruption and the development of a serious psoriatic erythrodermia. Alcohol should be forbidden.

Chronic Cases Old localized patches require more vigorous treatment than the generalized patches. If no improvement occurs at the end of two weeks, the ointment should be changed. Before ointment applications, the patient should take a hot bath and remove the scales with tincture of green soap and a brush. When ointments are used on the scalp a rubber bathing cap should be worn to prevent soiling the pillow. Since women will rarely permit the use of greasy ointments on the scalp they should be urged to follow instructions but to remove the medication after 1 hour by shampooing. The following drugs are best used in ointment form.

Precipitated sulfur (5 to 10 per cent) is helpful in treating the sensitive areas.

Precipitate sulfur	3-10
Ac. salicylic	5
Carbowax 1500 q.s. ad	100
M Ft. ung.	
Apply 1 bedtime	

Dioxyanthranol (Anthralin) may be used as a substitute for chrysarobin when the lesions are few and not too infiltrated. The drug does not have the irritating properties of chrysarobin. It is prescribed in 0.25 to 0.5 per cent strength.

For stubborn patches on covered areas chrysarobin ointment may be used. In the beginning it should be diluted 25 per cent with white petrolatum until tolerance is established.

Chrysarobin	2-10
Salicylic acid	6
Green soap	36
Petroleum q.s. ad	100

Riscol Mercuro and Siroil may improve the eruption temporarily when other drugs have failed.

Ammoniated mercury is also useful. A 5 to 10 per cent strength is used in cases of scalp involvement and in chronic patches. For the face, the trunk, the flexural folds and the genitals it is not advisable to use ointments stronger than 3 per cent because of the possibility of irritation.

Ammoniated mercury	3.0
Ac. salicylic	1.0
Petroleum q.s. ad	100.0

Tar is the safest drug used in the treatment of psoriasis. Its action is keratoplastic, antiacanthotic, vasoconstrictive and antipruritic. Crude coal tar a complex mixture of 48 per cent hydrocarbons (benzols, phenols cresols naphthalenes etc.) and pitch, is the most active therapeutically of all the tars. It sensitizes the skin to light because of its acridine content, which enhances the therapeutic effect. Tar is not used over hairy areas because of the danger of folliculitis. A popular form of therapy consists of using the following ointment according to a routine technic.

Crude coal tar	1 to 5
Zinc oxide	25
Petroleum q.s. ad	100

Crude Coal Tar—Ultraviolet Therapy
(after Goeckerman)

1. Patient takes a hot bath at bedtime with laundry soap and scrubs lesions with a brush until all scales have been removed.
2. Then a 3 per cent crude coal tar ointment is applied vigorously to the affected parts and a layer $\frac{1}{4}$ of an inch thick is permitted to remain. Wearing old pajamas, he then goes to bed.
3. In the morning he omits the bath but wipes off the remaining salve.
4. He then is given a general mercury-quartz light treatment in daily increasing doses.
5. This daily regimen is continued for about three weeks until the patient is free of lesions.

The above routine may be modified using 5 per cent crude coal tar in chloroform. This is painted on the lesions with a camel's-hair brush.

In extensive cases two types of baths may be used to soften the scales before applying local therapy: (1) the soap bath,

should be changed and a bland ointment or soothing lotion applied for a few days. Close supervision is therefore necessary in all cases under active therapy. It is best to respect the wishes of the patient as regards the therapeutic program.

Acute Cases Burow's zinc emulsion or Nivea oil should be used in treating acute cases. Astringent applications such as calamine lotions or zinc oxide-starch mixtures, also are useful.

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Magma bentonite	40.0
Lime water q.s. ad	100.0

Bed rest, sedation, autohemotherapy and colloid baths are often necessary to obtain relief. Stimulating local therapy is contraindicated to avoid the possibility of spreading of the eruption and the development of a serious psoriatic erythrodermia. Alcohol should be forbidden.

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Ac. salicylic	3
Carbowax 1500 q.s. d	100
M. Ft. ung.	
Appl. 1 bedtime	

Dioxyanthranol (Anthralin) may be used as a substitute for chrysarobin when the lesions are few and not too infiltrated. The drug does not have the irritating properties of chrysarobin. It is prescribed in 0.25 to 0.5 per cent strengths.

For stubborn patches on covered areas, chrysarobin ointment may be used. In the beginning it should be diluted 25 per cent with white petrolatum until tolerance is established.

rences are the rule. Most patients seek treatment for an extension of the disease or to obtain relief in persistent cases. The acute types usually are hospitalized, although extensive chronic types with or without an associated arthritis also improve more rapidly with hospital care, regulated diet and special technique.

The acute cases require bed rest, lotions, sedation, because of the severe itching and colloid baths. Alkalinization also may be necessary. When the acute stages have passed petroleum jelly will be tolerated when other ointments may cause a flare-up of the eruption.

Extensive chronic cases are treated with the combined coal tar ultraviolet light technic. The coal tar ointment is rubbed thoroughly into the patches at bedtime and removed in the morning with light mineral oil leaving a thin layer of the tar. The body then is divided into six areas, each receiving an increasing dose of ultraviolet light daily beginning with one minute at a distance of 30 inches. Following the light exposures the patient is instructed to bathe in a warm bath containing a coal-tar soapy solution and to remove as many scales as possible. Autohemotherapy and a low fat diet often are added to this regimen.

If results are not obtained with this therapy chrysarobin ointments are used. This drug stains the skin and the clothing and is irritating to the eyes. It must never be used on the face or the scalp or in the presence of nephritis. Wax paper should be used to cover the dressing in order to avoid soiling the bedclothes and irritating the uninvolved skin.

The Toxic Bullous Diseases

DERMATITIS HERPETIFORMIS
HERPES GESTATIONIS
PEMPHIGUS

ERYTHEMA MULTIFORME
BULLOSUM
HYDROA VACCINIFORME
NURSING ASPECTS

THESE diseases represent the direct effects of various etiologic agents upon the walls of the capillaries in the cutis resulting in localized edema. Bullae usually arise from an already edematous space resulting from a vasculitis.

The fluid in the vesicles or bullae contains coagulable serum, leukocytes, eosinophils and epithelial debris. After spontaneous or artificial rupture the bullous lesions are replaced by crusts, denuded areas or ulceration.

Bullous eruptions include the following diseases which must be differentiated by clinical studies observation and biopsies

Bullae with erythema

External causes—burns caustics, irritating ointments, extreme cold sunburn or ultraviolet light burns plant dermatitis, bullous impetigo

Internal causes—drug eruptions (iodide sulfa drugs, anti-pyrene) congenital syphilis, dermatitis herpetiformis erythema multiforme bullous lichen planus, hydroa aestivale

Bullae without erythema

Internal causes—pemphigus epidermolysis bullosa

The important generalized bullous eruptions are discussed here including dermatitis herpetiformis pemphigus and bullous erythema multiforme

DERMATITIS HERPETIFORMIS

Dermatitis herpetiformis (multiformis) or Duhring's disease is a chronic pruritic recurrent eruption characterized by polymorphous lesions with a tendency to group formation. The eruption may consist of papules vesicles bullae wheals and



FIG. 44. *Dermatitis herpetiformis*. Disease responded to Fowler's solution administered for a period of 3 months.

pustules. Most of the cases are of the papulovesicular variety. After the disease has been present for a considerable length of time and the patient has scratched constantly bullous lesions may continue to form but develop in the region of pigmented, crusted or erythematous patches.

The disease is characterized by intense itching pain, stinging and burning sensations resulting in nervous irritability. There is no impairment of health except nervous exhaustion. Periodic attacks occur at frequent intervals, so that the skin is never free of lesions. Although the disease is chronic, and the eruption is usually extensive the condition is not associated with mortality.

The eruption is generalized but shows a special predilection for the trunk, the buttocks, the groins, the shoulders, the posterior thighs and the knees. Although localized forms have been described the disease is more or less symmetrical.

The bullae vary in size and shape but are characteristically grouped, often irregular or stellate. In other cases the lesions are arranged in polycyclic designs or they may be grouped to form a crescent at the edge of an old erythematous patch.

Etiology. The cause of dermatitis herpetiformis is unknown. The following theories have been held: (1) bacterial or virus allergy (2) food allergy (3) hypersensitivity to iodides and bromides (4) endocrine imbalance and (5) psychosomatic factors.

Pathology. is characteristic in the vesicular and bullous types with vesicles at various levels in the epidermis. These are filled with fibrin eosinophils and other cells. In the upper cutis is an

Infiltrate of eosinophils and plasma cells, marked edema and dilatation of the capillaries and the lymph spaces.

Diagnosis. The diagnosis is based on the history of chronicity the presence of a symmetrical papulovesicular eruption, flare-up of the eruption following intravenous iodide marked itching and burning favorable response to sulfapyridine in many cases eosinophils refractoriness to treatment and the absence of any deleterious effect on the general health.

Cytodiagnostic examination of smears from the floor of the vesicles or bullae reveals numerous eosinophils a few PMN's and a few epidermal cells which retain their prickles and normal nuclei. The pemphigus cell is acantholytic and loses its prickles.*

Differential Diagnosis. Some cases may bear a superficial resemblance to scabies which should respond to the therapeutic test with Kwell Ointment when properly used. The common variety of the disease does not offer diagnostic difficulties, but bullous types occasionally are seen which cannot be classified without repeated observation and laboratory tests. The essential characteristics of the two diseases most likely to be confused with dermatitis herpetiformis are listed below

	<i>Dermatitis Herpetiformis</i>	<i>Pemphigus</i>	<i>Erythema Multiforme Bullorum</i>
Onset	Gradual	Sudden	Sudden
Eruption	Grouping and polymorphism Bullae arise from inflamed skin	No characteristic grouping or polymorphism. Bullae arise from normal skin	Iris or circinate lesions common with zone of hyperemia
Symptoms	Intense pruritus. General health good	Gradual decline in health	History of recent systemic infection. General febrile symptoms
Eosinophils	High (but not constant)	Present	Present
Cytologic smears	Normal epidermal cells	Acantholytic cells	PMN predominate
Prognosis	Chronic	Acute cases fatal	Recovery within 3 months

*See Winer, L. H. and Lipschultz, C. E. Comparative study of histology and cytology in vesiculating eruptions, *AMA Arch. Dermat. & S.* 63: 270-290, 1952

Prognosis The disease is incurable but can be controlled usually by the judicious use of sulfapyridine therapy. Remissions and exacerbations are common. The prognosis is much better in children for no known reason.

Eyster and Kierland found that the disease becomes quiescent or disappears spontaneously after 10 years in a majority of the cases.

Treatment. The disease is notoriously rebellious to treatment. All measures give only temporary improvement.

THE GENERAL TREATMENT consist of complete rest in bed during attacks and the examination for primary and secondary foci of infection. Potassium permanganate or colloidal baths relieve the itching temporarily. A high caloric and vitamin diet should be prescribed.

Sulfapyridine is the drug of choice but should be administered with caution. After the disease is controlled maintenance doses should be prescribed to prevent relapse with blood and urine studies at 2 week intervals. Often antihistaminics are useful in controlling the pruritus.

Promacetin, Diazone or hydrocortisone may control cases resistant to sulfapyridine.

LOCAL THERAPY is usually necessary to control the itching which is relieved by astringent lotions containing menthol, camphor, phenol or liquor carbonis detergens, even though the effects are transitory. Ointments containing 3 per cent sulfur are ordinarily well tolerated and seem to modify the eruption. When the disease is limited to small areas, the parts may be painted with a 1 per cent alcoholic solution of acriflavin.

HERPES GESTATIONIS

Herpes gestationis, a rare bullous disease, is probably a variety of dermatitis herpetiformis occurring in pregnancy. The disease usually begins after the third month of pregnancy and spontaneously disappears after term. The bullae are larger than those in dermatitis herpetiformis and the eruption progresses with the stage of pregnancy. Along with the generalized eruption, often fever, albuminuria, tetany and neuralgic pains are present. Severe cases may be followed by miscarriages or stillbirths.

Etiology is unknown but anti-Rh factors, liver damage, toxins or gonadotropic substances may play a part. The disease has been known to occur in patients having chorioepithelioma.

Prognosis. Dramatic improvement usually occurs after delivery.

Treatment is the same as that for dermatitis herpetiformis. Consultation should be had with an obstetrician to determine the necessity for interference with the pregnancy. Progesterone (50 to 100 mg daily) at regular intervals may be effective in some cases although hydrocortisone or ACTH therapy should be given a trial first.

PEMPHIGUS

This is a rare acute or chronic usually fatal disease characterized by a generalized eruption of bullae which appear on a normal skin. The benign cases those that get well are probably pemphigoid eruptions and not true pemphigus.¹ Occasionally an atypical eruption makes accurate diagnosis difficult. Attempts have been made to explain these variations by the existence of mutations with dermatitis herpetiformis and erythema multiforme bullosa but this theory has not been generally accepted.

Etiology. The cause is unknown. The following theories have been advanced: (1) the infectious theory (Welsh's streptococcus and Urbach's virus) is not widely accepted, (2) vitamin deficiency and (3) adrenal insufficiency with decreased chloride excretion and water retention.

The disease may occur at any age although it is more common in the 40 to 60 age group. There is no evidence that the disease is contagious or infectious.²

Clinical Types. The various types of pemphigus differ in their clinical picture, onset and course.

ACUTE FEBRILE PEMPFIGUS is characterized by a sudden onset and a rapid febrile course with the development of a generalized eruption of flaccid or tense serous hemorrhagic or purulent bullae developing on a normal skin. In some cases the disease may begin in the mouth with the development of painful lesions which rupture easily leaving denuded areas and ulcers. These interfere with mastication and swallowing. Within a week or longer inanition follows with death from sepsis or bronchopneumonia.

1 Rook, A. and Waddington, E. Pemphigus and pemphigoid, *Brit J Dermat* 65 425 1951.

2 For a more detailed discussion of the etiology and diagnostic problems, see Senior, F. E. Chronic pemphigus. *Trans. A.S.A. Arch Dermat & Syph* 65 429 1952.

Pathology Lever's studies¹ indicate that the bullae begin as a localized intercellular edema within and above the basal layer with gradual loss of the intercellular bridges. Lateral pressure causes cleft formation. Unilocular bullae, which contain eosinophils, are found in the epidermis. The corium also is edematous and occupied by a cellular infiltrate of eosinophils and small round cells.

Differential Diagnosis. Bullous drug eruptions (iodides, sulfa drugs and antipyrine) bullous erythema multiforme and the Stevens-Johnson syndrome may simulate acute pemphigus.



FIG. 45 Early pemphigus with involvement of mucous membranes. Note bulla on tongue. Fatal case, with death resulting from embolism in left iliac artery.

CIRCUMSCRIBED PEMPHIGUS VULGARIS is the common variety and consists of a generalized eruption of pea-sized to palm-sized bullae. The eruption which starts on the trunk or the extremities, gradually increases in extent until the entire body is involved. In the early stages the lesions sometimes are arranged in a cir-

¹ Pemphigus, histopathologic study. A.M.A. Arch. Dermat. & Syph. 64: 727-753, 1951.

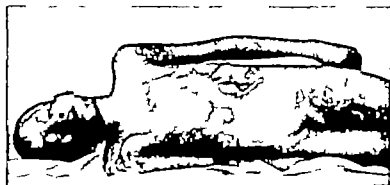


FIG. 46 Acute septic febrile pemphigus postmortem. (Dr. Leon Goldman's case)

climate or gyrate pattern. Early rupture of the bullae leaves large denuded areas, without any tendency toward epithelization especially in the axillary and the inguinal regions.

The normal skin easily separates upon slight pinching, friction or traction (Nikolski's sign), which is characteristic of the disease. In untreated cases, the ruptured bullae may be covered with thick, greenish crusts. After the disease has progressed for a variable length of time the skin develops a light or a dark brown pigmentation which may resemble that of Addison's disease.

The mucous membranes of the eyes or the mouth may be involved in the early or the late stages of the disease.

All cases of pemphigus are characterized by an offensive odor which is often diagnostic. Extreme tenderness may or may not be present but in all cases there is mental irritability as well as sensitivity to cold. The systemic symptoms are caused by a loss of plasma proteins and electrolytes as a result of the rupture of large bullous areas with circulatory collapse and eventual death.

Complications may develop including bed sores, thrombosis, stomatitis and conjunctivitis. Abscesses may occur in the vegetans type.

PEMPHIGUS FOLIACEUS This rare variety is characterized by patches of very flaccid bullae, yellowish-brown crusts and exfoliation of the edematous epidermis. It may appear *de novo* or develop from a pre-existing pemphigus vulgaris. In the typi



FIG 47 Chronic pemphigus, showing separation of stratum corneum, acantholysis and bullae formation.

cal cases the denuded areas have leaflike, upturned edges. In other cases the skin may present the picture of exfoliative dermatitis with scattered bullae and pigmented areas here and there. A characteristic offensive odor is present, due to decomposition of the exudate. The prominent symptoms in this type are extreme burning and soreness. After several weeks shedding of the hair and the nails occurs from trophic changes in the skin. Furunculosis and conjunctivitis are common complications.

PEMPHIGUS VEGETANS consist of localized denuded areas which undergo proliferative changes with the development of vegetating papillomatous masses covered with yellowish crusts and a progressive course without remissions. The presence of unruptured bullae elsewhere on the skin or on the borders of the vegetating growths is of diagnostic importance. When the vegetations involve large areas the temperature usually is elevated, and the patient is toxic or moribund. The vegetation growths have a special predilection for the anogenital the umbilical and the axil-



FIG. 48 Acute pemphigus with fatal termination (Dr. Leon Goldman's case)

lary regions. The foliaceous and vegetans types are better controlled with sulfonamides and chlortetracycline or chloramphenicol rather than the steroids.*

PEMPHIGUS ERYTHEMATOSUS (SENEAR USHER TYPE) is a benign form of pemphigus and runs a chronic course without affecting the general constitution although some cases eventually are transformed into pemphigus vulgaris. The condition which is characterized by bullae and crusts, simulates the clinical features of seborrheic dermatitis, pemphigus and bullous lupus erythematosus. The face and the trunk are affected usually and in some cases the mucous membranes also are involved. The treatment is similar to that of pemphigus.

OCULAR PEMPHIGUS is a form of mucous-membrane pemphigus which involves the conjunctivae resulting in cicatricial changes and eventually blindness. The disease may appear first in the mouth and affect the eyes at a later period. If the skin is involved the bullae occur late in the course of the disease usually localized to certain areas and rarely generalized. Other mucous membranes may be affected. The course of the disease is benign, slow and chronic the eye lesions resistant to therapy with cicatrization the usual end result. The general health rarely is impaired, and the disease usually is limited to the eyes.

Differential Diagnosis. Pemphigus vulgaris must be differentiated mainly from dermatitis herpetiformis and from erythema multiforme. In pemphigus, there is activity all the time,

the bullae arise from normal skin, and the eosinophil count is increased only slightly. In dermatitis herpetiformis, the lesions are polymorphous, the mouth is involved only rarely, pruritus is more intense, relapses are common, and eosinophilia is high (5 to 25 per cent). In erythema multiforme bullosum the bullae arise from bluish-red inflammatory areas, a history of sore throat, febrile disease or drugs often is obtained and the general health of the patient rarely is affected. In impetigo contagiosa the bullae are localized, crusting appears rapidly and the response to specific therapy is rapid.

Cytologic smears, when used in conjunction with a thorough clinical examination, are helpful in separating pemphigus from the other bullous diseases.

From chemical examinations of the blood Fisher* was unable to arrive at any significant conclusions regarding their diagnostic value in pemphigus.

Pemphigus vegetans may be mistaken for pyoderma vegetans, vegetating bromide or iodemia or condylomata. In pemphigus bullae are found elsewhere, progression of the eruption is the rule and a fatal outcome is inevitable.

Diagnosis. Pemphigus should be suspected in all cases in which there is a history of unruptured bullae developing suddenly on the normal skin or mucous membranes. The presence of systemic disturbance, mouth lesions, continuous activity, loss of weight and strength, low plasma proteins, secondary anemia (which parallels the severity and the course of the disease) and a positive Nikolski's sign helps to corroborate the diagnosis. Microscopic sections offer suggestive but not positive evidence. Time often establishes the diagnosis in cases where differentiation is difficult.

Prognosis. In general, the prognosis of this serious disease has improved with the advent of steroid therapy. Remissions are longer and complications are fewer. However, 90 per cent of the cases result in death within 3 months or longer. The signs of remission are dry crusting of old lesions, failure of new lesions to develop, epithelialization of denuded areas and a brighter mental outlook.

In the acute cases a fatal termination may occur within a week or 10 days. The chronic cases are characterized by long remis-

*Fisher I. Pemphigus vulgaris. clinical and laboratory study. A.M.A. Arch. Dermat. & Syph. 66:49-58, 1952.

sions and exacerbations. Each succeeding attack leaves the patient weaker and weaker until death finally occurs from inanition, toxemia, lung or kidney complications.

Treatment. THE GENERAL TREATMENT consists of bed rest, good nursing, and a high-vitamin and caloric-concentrated diet to counteract the toxic effect of the disease on the general health. If the mouth is involved making mastication difficult, nasal feeding may be necessary. Anesthetic lozenges may be prescribed for the painful oral and throat lesions. The use of a cradle prevents the bed covers from irritating the skin. Plenty of blankets should be provided to protect the patient from chills. Tincture of opium (5 drops t.i.d.) should be used routinely to reduce the discomfort of the disease.

LOCAL TREATMENT has no effect on the eruption per se but medication is necessary to control the pain resulting from the ruptured bullae. This consists of the use of soothing ointments, lotions or powders although the limits of toleration vary. A 5 per cent tannic acid in tragacanth jelly is tolerated by some patients; others respond to a mixture of equal parts of olive oil and lime water or acriflavine 1:3,000 in petroleum gauze. In some cases, a corn starch powder bath or borated talcum-powder applications are sufficient to keep the skin dry and the patient comfortable. If the patient is not bedridden permanganate baths should be prescribed to counteract the fetid odor. Pressure bandages over eroded areas protect the skin from trauma, prevent secondary infection and stimulate epithelization.

ACTH, hydrocortisone and Prednisolone are the only drugs that appear to be effective. In some cases the results are dramatic, in others however they are a disappointment. If the disease responds to these drugs the proper dose must be determined to keep it under control and a maintenance dose prescribed. The patient should be under close observation to detect any untoward effects from long-continued steroid therapy.

Blood transfusions are indicated early to counteract the progressive anemia and the lowered resistance of the patient. If hypoproteinemia is present (plasma protein below 4 per cent) intravenous injections of plasma protein are useful to correct the tissue edema.

The type of therapy used depends on the duration of the disease, the extent of the eruption and the general condition of the patient. The following drugs and therapeutic measures may be useful at some stage of the disease:

1 ACTH. In acute fulminating cases, the intravenous route should be used until the eruption is controlled. The dose should be cut down as soon as feasible. The minimal maintenance dose varies in individual cases. To be effective the drug should produce a decided reduction in the eosinophil count. In the chronic cases, prednisolone is the drug of choice. The dose of cortisone (I.M.) is 300 mg. in divided doses the first day 200 mg. the second day and gradually tapered off to a maintenance dose of 50 mg. daily. A low-salt diet and the use of potassium chloride by mouth to keep the sodium and potassium blood levels within normal limits is important.

2 Arsenic. Acetasone. Carbarsone or Asiatic pill also may control the disease at various times and may be used between courses of the steroids.

3 High-protein intake or protein hydrolysates.

4 Vitamin K (if prothrombin levels are low) to lessen danger of thrombi.

5 Antibiotics (oxytetracycline, chlortetracycline or chloramphenicol) beginning with 250 mg. every 6 hours and gradually reducing the dose after 1 week, are erratic in their effect and certainly not as useful as ACTH or cortisone.

6 Testosterone (to restore positive nitrogen balance and to prevent osteoporosis)

7 Good and adequate nursing care.

ERYTHEMA MULTIFORME BULLOSUM

Erythema multiforme bullosum is an acute inflammatory disease characterized by an acute onset and a multiform eruption including large bullae surrounded by a red halo. The duration of the condition is relatively short, and the prognosis is good except in the extensive septic cases.

Clinical Signs. The striking feature is the symmetrical eruption of bluish-red macules, papules and bullae, which often appear in crops. When the bullae are closely examined they do not appear to arise from the normal skin. On the contrary they either are surrounded by a red halo or develop from the edge of an erythematous patch. The bullae which on rupture leave tender raw areas, are tense rather than flaccid and contain serous or hemorrhagic contents. In some cases the bullae are ringed or

circinate. The fading lesions may leave a zone of temporary pigmentation.

The anterior and the posterior trunk and the extensor surfaces of the extremities frequently are affected. Lesions also may occur on the lips in the mouth or on the conjunctiva.

Constitutional symptoms are often absent but when the disease is extensive fever, headache and arthritic pains may be present. Sore throat and tonsillitis are not uncommon in many of the cases.

Etiology. Erythema multiforme usually attacks young adults while pemphigus affects an older group. There is a definite seasonal incidence—most of the cases occur in the spring or the fall.

Types. (1) The idiopathic or recurrent. (2) symptomatic or secondary, which may be caused by the following: focal infection, intestinal toxins, idiosyncrasy to drugs and sera, acute infectious diseases including rheumatic fever, chronic visceral disease, and (3) the Stevens-Johnson syndrome (virus origin).*

Pathology consists of edema of the corium, dilatation of the blood vessels and a perivascular infiltration consisting of lymphocytes, polymorphonuclear leukocytes and red blood cells.

Differential diagnosis is discussed under Pemphigus.

Treatment. A thorough physical examination and blood-chemistry studies should be made in an attempt to find the cause. Extensive cases which may require prolonged observation should be hospitalized.

Supportive therapy includes a high vitamin diet, low salt intake, folic acid, nicotinic acid amide and the antihistaminic drugs. Rutin is useful in these cases to reduce the activity of hyaluronidase in the tissues and thus reduce the permeability of the cellular and the vascular structures.

Daily intravenous calcium gluconate injections and blood transfusions. ACTH and hydrocortisone in the severe cases, are useful. Aureomycin, Terramycin or Achromycin (250 mg q 4 h) may be of service in cases with an infectious basis. Local treatment consists of the use of astringent lotions and colloidal baths.

Prognosis. The average case clears up within a month. Recurrences are not infrequent unless the cause is discovered and removed.

* See Womack, C. R. and Randall, C. C. Erythema multiforme exfoliatum, its association with viral infections, *Am J Med* 15:613 1953.

STEVENS-JOHNSON SYNDROME

The rare Stevens-Johnson syndrome a fulminating form of bullous erythema multiforme, is characterized by sudden onset, symptoms of an acute respiratory infection septic fever purpura, prostration, a generalized bullous eruption on a edematous-erythematous skin early involvement of the mucous membranes, including the ocular (which may be serious) and mouth lesions which may interfere with feeding. This type runs a course of 2 or 3 weeks, but the mortality rate is about 20 per cent.

Treatment consists of hospitalization hydrocortisone or ACTH given early in adequate doses, a trial of the broad-spectrum antibiotics, ophthalmologic consultation in eye involvement, blood transfusions early and good nursing care



FIG. 49 The Stevens-Johnson syndrome with extensive erythema multiforme bullosum eruption. (From Dr. John C. Slaughter)

HYDROA VACCINIFORME

Hydroa vacciniforme (aestivale) is a rare recurrent disorder in children resulting from an abnormal sensitivity to sunlight, and is characterized by an eruption of vesicles and bullae on the face and the exposed parts. The disorder usually begins in infancy or later and gradually disappears after puberty.

The eruption consists of vesicles, transparent bullae, excoriations and brownish crusts. After repeated attacks, the sites of the old lesions are occupied by depressed varioliform scars. Itching may occur at the onset of attacks.

The condition recurs each spring reaches its height during July and August and then gradually fades.

Chiefly the cheeks, the forehead, the ears and the extensor surfaces of the arms and the forearms are involved. The general health is otherwise good.

Etiology The condition is due to a congenital hypersensitivity to the ultraviolet rays of the light spectrum. A disturbance of porphyrin metabolism is apparently not a factor although a few cases have been reported in which congenital porphyruria was present. The condition also has been ascribed to a disturbance of the sulfur metabolism (cystine absorbs ultraviolet light). The absorption of photodynamic toxins from streptococci in the intestinal tract is another factor worth further investigation.

Prophylaxis. Direct exposure to the sun should be avoided. Ointments containing 15 per cent β -aminobenzoic acid supply sufficient protection from the harmful rays.

Treatment consists of soothing lotions or ointments, and the empiric use of nicotinic acid amide, liver extract or estrogens to reduce hypersensitivity. A course of Aralen, which may act as a light barrier is worth a trial. The drug is given 3 times daily the first week, twice daily the second week and once daily as a maintenance dose. Close supervision is necessary to detect toxic manifestations.

NURSING ASPECTS

The bullous dermatoses require hospitalization, specialized nursing care, adequate laboratory examinations and special therapeutic measures. The bullous drug eruptions and bullous erythema multiforme usually clear in a few weeks but pemphigus may require months of hospital care.

Erythema Multiforme Bullosum. Colloid or permanganate baths are given daily. All bullae or blisters should be opened daily with a sterile hypodermic needle and the prescribed lotion then should be applied with sterile gauze. A biopsy set and sterile slides for smears from the blister fluid should be available. The diet should be soft if mouth lesions or lip lesions are present, and attention should be given to the hygiene of the mouth and the eye.

Pemphigus. Most nurses shudder at the thought of a new pemphigus case to be hospitalized because of the time-consuming care that this disease requires. Kindness, sympathy and reassurance are necessary on the part of the nurses assigned to care for these unfortunate patients. Although rubber gloves should be worn when bathing the patient or applying medication there is no danger of infection. To overcome the foul odor in advanced cases, permanganate dressings or permanganate baths are prescribed. Since the blisters rupture easily leaving painful erosions, petroleum-jelly dressings or pressure bandages are employed. Infected bullae should be opened gently with a sterile hypodermic needle, and 2 per cent gentian violet then should be applied. ACTH should be available for intravenous drip or injection.

The Pyodermas

DISEASES DUE TO STREPTOCOCCI	ACNE CONGLOBATA
IMPETIGO CONTAGIOSA	PYODERMA VEGETANS
IMPETIGO IN INFANTS	FOLLICULITIS KELOIDALIS
ECTHYMA	ACNE NECROTICA MILLIARIA OF THE SCALP
PYODERMA GANGRENOSUM	FOLLICULITIS ABSCEDENS ET SUFFODIENS
ERYSIPELAS	PYODERMA FACIALE
RECURRENT CELLULITIS	THE ACRODERMATOSES
ERYSIPELOID	ACRODERMATITIS CONTINUA (HALLOPEAU)
DISEASE DUE TO STAPHYLOCOCCI	FISTULAR BACTERID
SUPERFICIAL PUSTULAR FOLLICULITIS	NURSING ASPECTS
FURUNCULOSIS	
SYCOSIS VULGARIS	

THE pyodermas comprise the largest group of skin diseases. These pyogenic infections may be simple (impetigo) complex (pyoderma gangrenosum) primary secondary associated with bacterial sensitization superficial or deep. Some may respond to local anti bacterial therapy others require systemic treatment.*

The normal flora of the skin consists of a diversity of resident and transient bacteria which include the nonhemolytic and the hemolytic *Staphylococcus aureus* and *albus* micrococci diphtheroid bacilli and *B. pyocyaneus*. So long as the resistance of the host is maintained at a sufficiently high level, the bacteria constituting the normal flora do no harm. The pyodermas include a large group of localized and generalized eruptions. Simple dermatoses may be secondarily infected. Prolonged bacterial infections, if improperly treated may result in an indolent chronic eczematous dermatitis as a result of local bacterial sensitization and the effects of trauma from scratching.

*For an excellent review of the therapy of the pyodermas see Lhlogood, C. J. and Mullins, J. F. Management of bacterial infections of the skin. Postgrad Med 12 15 1952

The clinical type of infection produced by these bacteria depends on individual susceptibility local factors of immunity strain of the organism, general and local hygiene, age of the patient and anatomic factors (halry areas and regions rich in sebaceous glands being more susceptible) production of exotoxins (may cause necrosis) development of local or general sensitization and climatic conditions affecting the soil.

Staphylococci. The various strains of staphylococci, of which there are many differ greatly in virulence. The body response to this organism is fairly uniform, and the clinical manifestations usually typical. The *Staphylococcus aureus* which is the most virulent is found in the normal flora of the skin the nasal passages and the dust in the air. *Staphylococcus albus* and *Staphylococcus citreus* are rarely pathogenic they are present in certain banal lesions and are part of the normal flora of the skin.

There is no doubt that the skin is a more suitable soil for the growth of staphylococci than for streptococci. The former are normally present in warm moist areas, about the hair follicles and in the large sebaceous openings. The clinical lesions produced by the staphylococcus depend on the virulence of the strain the patient's resistance and the region affected.

The streptococci usually produce more extensive lesions and a systemic response in the form of fever chills and leukocytosis. They are classified as *Streptococcus hemolyticus* *Str viridens* and *Str nonhemolyticus*. The hemolytic type is as a rule more virulent than the nonhemolytic types.

Members of the hemolytic group are *Str erysipelatis* (erysipelas) *Str scarlatinae* and *Str pyogenes* (wound infection).

Little is known about the virulence of the various strains of streptococci. They are never saprophytic but are indicative of an infective focus somewhere on the surface. Some are apparently harmless while others may bring about death in a few hours. In any event, any disturbance of the defenses of the tissues may result in a serious streptococcal septicemia.

BACTERIAL ALLERGY. Skin testing with bacterial antigens is not useful in dermatologic diagnosis for the following reasons (1) multiplicity of strains, (2) previous exposure to organisms in health and disease (3) nonspecificity of antigens (commercial) and (4) complex antigenic composition of bacteria. The therapeutic or the surgical removal of foci is a more definite approach.

SENSITIVITY ANTIBIOTIC TESTS. Since many strains of staphylococci and streptococci may vary widely in their susceptibility to antibiotics these tests should be made in all chronic relapsing infections, in extensive cases and in those cases where a lack of response suggests drug resistance or a change in the infecting organism. However there is often a lack of correspondence between the in vitro test and the in vivo clinical response so that a change in antibiotics is necessary if results are not satisfactory after a 2 week trial.

CULTURAL STUDIES Positive cultures obtained from pyogenic lesions are not necessarily proof of the etiology they may indicate secondary infection or may represent the normal flora of the skin. A positive skin test with a weak dilution of the organism (1:100,000 to 1:1,000,000) associated with a flare-up of the eruption is more suggestive of a bacterial factor.

Chemotherapy of the Pyodermas. The sulfonamides are not safe drugs to use in dermatologic conditions because of their high index of sensitization when prescribed for local or systemic disease. In serious conditions, however e.g. actinomycosis, a sulfonamide may be combined advantageously with antibiotic therapy.

DRUG FASTNESS. Bacteria may become adapted to the action of bacteriostatic and antibacterial drugs. This tendency is increasing annually so that we may safely presume that 50 per cent of all pyodermas are resistant to penicillin, 30 per cent to oxytetracycline and 25 per cent to chlortetracycline. Therefore it is important to change to other antibiotics if clinical results are not satisfactory. In serious infections in vitro laboratory tests should be made before treatment is instituted.

Criteria for the Evaluation of Topical Antibacterial Agents Used in the Treatment of the Pyodermas. Since the physician is confronted with a large number of antibiotics and antiseptics the following facts should be considered in evaluating these drugs: (1) wide spectrum of effectiveness (2) low sensitizing potential, (3) low systemic toxicity (4) minimal tendency to development of resistant strains after prolonged topical use (5) drug not used for systemic administration (6) no tendency to development of monilial infections at site of application (7) patient acceptance and (8) reasonable cost. These criteria are proposed by Salzberger and Baer (*Treatment of Pyodermas Year Book of Dermatology and Syphilology 1950 pp 952*) in an excellent review of the subject.

Choice of Topical Antibacterial Drugs.

DYES. Tr. Iodine Mercurochrome Mercresin, Metaphen Zephiran etc.—not to be used for local treatment. Use limited to preparing site for minor surgery.

HOT BOWTIE PACKS. Useful for increasing local immunity loosening pyogenic crusts and reducing pain.

ADMONIOLATED MERCURY In a 3 per cent ointment, useful in Impetigo but not in infected eczemas.

VIOGON OINTMENT (or cream) Useful in superficial pyoderma.

SULFONAMIDE OINTMENTS High index of sensitization prohibits their use.

PENICILLIN OINTMENT Too many local reactions for routine use.

BACITRACIN OINTMENT Best for routine use as a broad-spectrum antibiotic.

TERRAMYCIN NEOMYCIN AUREOMYCIN ERYTHROMYCIN ACTHOMYCIN CHLORAMPHENICOL, TETRACYCLINE OINTMENTS. Useful in hemolytic staphylococcal infections.

AUREOMYCIN TERRAMYCIN BACITRACIN AND CHLORAMPHENICOL, OINTMENTS Used for hemolytic streptococcal infections.

COMBINED ANTIBIOTIC OINTMENTS Probably no advantage.

POLYMYXIN B OINTMENT Pseudomonas infections.

NEOMYCIN AND CHLORAMPHENICOL OINTMENTS. B. proteus infections.

TYROXIN CREAM Safe but bactericidal effect too weak.

DISEASES DUE TO STREPTOCOCCI**IMPETIGO CONTAGIOSA**

An infectious and contagious disease involving the superficial layers of the epidermis resulting in the formation of vesicles which rupture readily. It is one of the commonest diseases and one of the easiest to cure.

Clinical Description. The primary lesion is a red macule which rapidly becomes a flaccid vesicle. Following central rupture and peripheral extension it soon becomes a pustule. The seropurulent exudate dries up, leaving thick honey-colored crusts which have a "stuck on" appearance. Removal of the crust discloses a superficial ulcer with thin overhanging edges of epidermis. When the lesions are fully developed they vary in size

from 0.5 to 4 cm. They have a tendency to be round or oval in shape occasionally they coalesce to form serpiginous shapes. New lesions appear rapidly within a few days by autoinoculation.

Although the face usually is affected, the nostrils, the chin, the ears, the nail folds, the hands and the buttocks also may be involved. Impetigo is often associated with pediculosis of the scalp. In other cases it is produced by constant scratching in such diseases as eczema, millaria, insect bites, purulent rhinitis and scabies.

Etiology. The disease is caused primarily by the *Str. pyogenes* which is found in large numbers in the vesicles. However the vesicle rapidly becomes infected with staphylococci and ruptures with the formation of a crust. Impetigo commonly is spread by the hands and contaminated towels. Over 90 per cent of the cases occur during the summer months. The infection may occur at any age and in both sexes but is commonest in children. Mild trauma is a predisposing cause.

Varieties. The common type *Impetigo vulgaris* has just been described. The bullous type *impetigo contagiosa bullosa*, which is more unusual is a prolongation of the vesicular stage. A third variety is characterized by the presence of circinate crusted patches (*Impetigo circinata*). This variety often is mistaken for ringworm but the acuteness of the eruption, the presence of pustules, the rapid response to therapy and the absence of fungi help to establish the correct diagnosis. A fissured type occurs at the corners of the mouth, behind the ears and under the breasts.

Complications. Paronychia often is seen as a complication when impetigo is present on the hands. Furunculosis commonly occurs in neglected children and infants. In Negroes suppurative adenitis may follow the more severe types. Acute glomerulonephritis may occur in rare cases both as a result of the infection and from absorption of ammoniated mercury ointments used in the therapy.

Pathology. Impetigo is a superficial epidermal lesion consisting of a bulla, a vesicopustule or a crust. The bulla lies between the prickle-cell layer and the stratum corneum. It is filled with serum, leukocytic debris, streptococci and staphylococci. The lymph spaces and the capillaries in the papillae are dilated.

Prognosis. If untreated impetigo spreads and heals until most of the cutaneous surface is covered. Since the infection is limited to the epidermis scarring does not occur.

Treatment. The first principle of treatment in impetigo is to remove the crusts to permit penetration of the medication. This can be accomplished by physiologic saline warm boric packs 1 5,000 permanganate or equal parts of hydrogen peroxide and water. Gentle washing of the involved areas with soap and water daily is important from the standpoint of antiseptics.

EXPOSED AREAS. In the average case bacitracin ointment (500-1,000 units per Gm.) or 3 per cent ammoniated mercury ointment applied to each and every lesion results in a cure within a week. They should not be used for longer than 7 days, and the usual precautions observed. In resistant cases half strength Quinolor Ointment (Squibb) or a trial with the various antibiotic salves is advisable. A dressing is not required. The surrounding unaffected parts should be sponged daily with a saturated solution of boric acid as a prophylactic measure. Since the denuded epidermis is sensitive, strong antiseptics do more harm than good aggravate the infection and spread the disease.

COVERED AREA. Other methods of treatment may be employed if the covered areas are involved. Dressings should be used to prevent irritation by the clothing. In many cases a 2 per cent alcoholic solution of gentian violet hastens resolution. This drug has a specific effect against the streptococcus and is not irritating to the denuded epidermis. The only objection is the color which can be removed with soap and water. An equally good application is a 5 per cent solution of silver nitrate in sweet spirits of nitre. In generalized cases ultraviolet light baths seem to improve the general resistance of the skin. In extensive cases penicillin by injection is the drug of choice unless there is a history of sensitization when achromycin may be used.

IMPETIGO IN INFANTS

Impetigo in infants is caused by the *Staphylococcus pyogenes aureus*. The lesions are not present at birth which helps to differentiate the disease from the bullous syphilide. Epidemics occasionally occur in the nurseries of hospitals. Usually the eruption is bullous in infants, often involving the scalp the axillary spaces, the chest and the anterior surface of the neck. Some cases complicate miliaria and infantile eczema.

Treatment. Even a weak ammoniated mercury ointment is usually too irritating for the skin of an infant and, if used over extensive areas, may produce mercurial intoxication. Lotions con-

taining 3 per cent Vioform are superior to ointments when the neck or the body folds are affected. It is advisable to snip the roofs of the vesicles with sterile scissors and to bathe the patient once a day in a 1:5000 permanganate bath. The lesions should be painted daily with a 2 per cent aqueous solution of gentian violet or a 5 per cent silver nitrate solution. The arms of the infant should be splinted to prevent reinoculation. The average case responds to antibiotic creams. To avoid sensitization, there is a practical advantage in using effective topical antibiotics that are not in use systemically (bacitracin-neomycin).

Prophylaxis. In cases originating in nurseries the ward should be closed to new arrivals until all the cases are discharged, and the rooms disinfected. This usually requires about two weeks. Infected cases should be isolated and treated by a special personnel who should not be required to care for uninfected cases. Doctors, nurses, attendants and laundry personnel may be carriers.

Prevention of Impetigo in Nurseries

1 Remove at birth all excess vernix caseosa and blood from face and folds with sterile olive or cottonseed oil.

2 Bath Routine. The head and the neck, the arms, the trunk and the legs and the buttocks are cleansed with a germicidal soap (PHISOX (Winthrop) or Septisol (Vestal)). Then the infant is placed in a warm bath, rinsed well in clear water and dried completely. The skin must not be irritated or traumatized.

3 The cord is cleansed with 60 per cent alcohol.

4 The mother's breasts are washed with soap and water each morning. She also washes her hands before each nursing.

5 The physician must wear sterile rubber gloves, mask and gown when handling the infant.

6 No one with pyogenic skin lesions should be admitted to the nursery. Children should not be admitted to maternity divisions.

7 Ultraviolet germicidal units should be used in nurseries to prevent cross infections.

ECTHYMA

Ecthyma (jungle rot) is a form of impetigo characterized by deep ulcerations covered with thick brownish crusts and associated

with lowered resistance and poor hygiene. Often there is a history of "picking a pimple." The disease commonly appears on the legs but may develop anywhere on the body. Since ecthyma is an ulcerative process, it leaves scars.

Pathology The inflammatory reaction extends downward to the upper corium, with the epidermis replaced by a crust containing streptococci. Healing results in scar formation with pigmentation at the periphery.

Differential Diagnosis. Primary tuberculous complex, extra genital chancre and the ecthymatous secondary syphilid must be considered.

Treatment. Local therapy is similar to that used in impetigo. General tonic treatment is advisable, such as iron and arsenic medication, vitamin A and ultraviolet light baths. Antibiotic ointments preferably bacitracin or 2 per cent gentian violet are applied locally.



FIG. 10. Impetigo contagiosa. Note "stock on" crusts.



FIG. 51 Echthyma. Lesions followed a secondarily infected scabies.

INFECTIOUS ECZEMATOID DERMATITIS

(See p. 72)

PYODERMA GANGRENOSUM

Pyoderma gangrenosum consists of cutaneous abscesses which break down forming large ulcers with undermined advancing borders. *Bacillus pyocyaneus* and staphylococci as well as streptococci are found in the lesions. Symptoms include fever, loss of weight and increase in number of skin lesions with flare-up of internal focus. The condition often is associated with empyema, dysentery and chronic ulcerative colitis, some cases of which are caused by a strain of streptococcus. Brunsting and his co-workers* regarded the disease as but one part of a generalized infectious syndrome characterized by a marked lowering of the bodily resistance to the invading organisms. There is evidence available that suggests that the lesions of pyoderma gangrenosum are embolic or produced by an Arthus phenomenon. Differential diagnosis is made from iododerma and bromoderma, generalized blastomycosis, erythema induratum and factitious dermatitis. The stools should be examined routinely for *E. histolytica* to rule out amebic infections.

Prognosis is guarded. Several months may be required for healing. Recurrences are not uncommon in patients with chronic ulcerative colitis.

* Brunsting L. A. et al. Pyoderma (Ecthyma) gangrenosum. *AMA Arch. Dermat. & Syph.* 22: 655, 1910.

Treatment consists of a general tonic regime, antibiotics, high-vitamin diet and blood transfusions in severe cases. Zinc peroxide paste or brilliant green and antibiotic ointments sometimes aid in the healing of small areas. Debridement to remove necrotic tissue is important.

ERYSIPELAS

Erysipelas is a localized acute inflammation of the skin and the subcutaneous tissues caused by the *Streptococcus erysipellatis* and characterized by fiery redness, edema and a raised indurated advancing border



FIG. 52 Erysipelas of right side of face secondary to impetigo of the eye

Clinical Description. The disease usually is ushered in with prodromal symptoms of malaise, chills and a fever of 100° to 105° F. In the severe cases there is early toxemia and prostration.

The infection is characterized by the presence of a well-demarcated, fiery-red area with or without edema and vesiculation. A characteristic sign is the advancing red, raised indurated border. As the disease progresses the older areas fade, turn brown and desquamate.

When the face is involved the disease often starts from a fissure in the nostril or the ear. At first unilateral it spreads until the entire face is affected. Edema of the eyes is a usual

complication. As the disease spreads upward it seems to be barricaded by the hairline and usually stops there. Recurrences are common.

Complications are more apt to occur in the very young and the aged postoperative cases and following extensive injuries. They consist of bronchopneumonia meningitis, septicemia and cardiac complications. In facial cases chronic lymphedema or cheilitis may follow recurrent attacks.

Etiology The exciting cause is the *Streptococcus erysipellus*. Facial cases often follow aural or nasal fissures or result from pulling hairs in these regions. However the disease may develop from a banal scratch or injury.



FIG. 53. Recurrent cellulitis of the ear.

Erysipelas may follow vaccination and varicose ulcers where hygiene has been neglected or irritating applications used. The disease often is seen as a complication of dermatophytosis of the feet, especially during the summer months.

Differential Diagnosis. Erysipelas must be differentiated from contact dermatitis (medication plants etc.) and acute solar dermatitis.

Pathology Sections show a marked vascular dilation of the corium with intense edema and dilated lymph channels containing streptococci lymphocytes and polymorphonuclear leukocytes.

Prognosis. Recovery is the rule within 10 days. However the disease may be fatal in the very young, aged, alcoholics and in those with low resistance from serious injuries.

Treatment. Severe cases should be hospitalized. Patients with a mild form of the disease should remain at home until all danger has passed. While the value of local applications is debatable, continuous ice-cold packs of saturated solutions of magnesium sulfate or 10 per cent aqueous solutions of ichthylol are comforting to the patient.

Penicillin by injection is the drug of choice. If there is a history of sensitization, one of the broad-spectrum antibiotics should be given orally (250 mg. q. 6 h.) until the patient is afebrile and the clinical signs have disappeared.

In the chronic recurring cases x-ray therapy, foreign-protein therapy, removal of focal infections or streptococcus vaccine may be beneficial.

RECURRENT CELLULITIS

Recurrent cellulitis (elephantiasis nostras or solid edema) is an infection of the deep tissues which may be secondary to a superficial streptococcal dermatitis. Occasionally it may follow erysipelas. The face, the ears and the lips usually are affected.

Prognosis is poor in the chronic cases if fibrosis is present, and the cause undetermined.

Treatment consists of the use of streptococcus vaccines or serum, penicillin or streptomycin injections for at least 2 weeks, foreign protein therapy, sulfonamide drugs. X-ray therapy may be useful in the resistant cases.

ERYSIPELOID

This condition which resembles a mild erysipelas consists of purplish bands, streaks or plaques usually occurring on the hands or the fingers of handlers of meats and vegetables, fishermen and fish dealers as a result of puncture wounds. There is an absence of vesiculation, adenopathy and lymphangitis. The incubation period is about one week. Since trauma plays an important part the disease is usually unilateral. Occasionally farmers or veterinarians may acquire the disease from pig bites. There are no constitutional symptoms. The organism responsible for this condition is the gram-positive *Erysipelothrix rhusiopathiae*, a funguslike organism.

Prognosis is good. The condition clears up with proper therapy within a week or 10 days.

Treatment. Penicillin by injection is standard therapy unless the patient is sensitive to the antibiotic, in which case oral Aureomycin or Terramycin should be used.*

Prophylaxis. The hands should be scrubbed with soap and water after wounds resulting from handling fish.

DISEASES DUE TO STAPHYLOCOCCI

SUPERFICIAL PUSTULAR FOLLICULITIS

This the impetigo of Bockhart is a coccogenic dermatitis characterized by an eruption of yellow pustules pierced by a hair. The scalp, the face and the neck are sites of predilection. Since the infection is limited to the upper third of the follicle it is a superficial pyoderma.

Etiology includes miliaria with secondary infection, the use of irritating ointments, e.g. tar or mercury, contact with oils or greases, close shaving or poor hygiene of the skin and the scalp. *Staphylococcus albus* usually can be isolated from the pus.

Treatment consists of packs of hot boric or 1:8 salicylic solution, a trial of the antibiotic ointments, Quinolone ointment and good hygiene. The use of ultraviolet light helps to raise the resistance of the skin. For prophylaxis one of the hexachlorophene soaps should be used.

FURUNCULOSIS

A furuncle is a localized acute inflammatory lesion beginning in a hair follicle about which an area of cellulitis develops. It usually terminates in suppuration and necrosis.

Clinical Description. A furuncle begins as a tender and painful folliculitis of pin-point size surrounded by an inflammatory areola. Unless the lesion is aborted there is increased tenderness, redness, heat and swelling resulting in a localized abscess which is pierced by a hair. Usually on the third day suppuration or pointing occurs and the boil is said to have "come to a head." During this stage there is a throbbing pain with limited movement of the affected parts. Sloughing or spontaneous termination of the process occurs on the eighth day unless complications de-

*For other forms of therapy consult Klauder, J. V. Erysipeloid as an occupational disease. JAMA 111:1345, 1918.

velop. The slough or "core" consists of a greenish yellow tenacious plug which may be tinged with blood.

Furuncles are usually single, but more than one may be present in adjacent areas or at widely scattered points (multiple furunculosis). Any part of the cutaneous surface may be the site for a furuncle but the common locations are the face the neck, the axillae, the buttocks, the trunk and the thighs. The duration of the furuncle varies from a few days to several weeks.

Constitutional symptoms consist of irritability in the acute types and secondary anemia or albuminuria in the chronic types. The usual sequelae consist of scars, the character of which depend on the extent and the severity of the infection and the treatment used.

Etiology Single furuncles usually are caused by trauma to the hair follicle. Neck lesions often develop from irritation from a frayed or dirty collar eyebrow lesions from plucking hairs nasal lesions from plucking the vibrissae buttock lesions from friction irritation and poor hygiene.

Multiple furuncles are produced by various external and internal factors. In cases where a single boil has been poulticed new lesions may result from reinoculation of the sodden epidermis. Multiple furunculosis is a common complication in dermatoses characterized by itching such as scabies, pediculosis, eczema, impetigo and prickly heat. Nasal infections with contamination of the skin from the fingers is a common cause of generalized furunculosis. In other cases, obstruction and infection of the hair follicle in occupational dermatoses due to oil greases and tars may produce furunculosis. In addition iodides and bromides may be responsible for acneform, pustular lesions resembling furuncles.

Furunculosis of the scalp in infants frequently results from poor hygiene, soiled pillows, excessive perspiration of the head lack of ventilation in the crib and "cradle-cap."

Bacteriology Furuncles usually are caused by the *Staphylococcus aureus*. Superficial banal infections of the hair follicles are occasionally due to the *Staphylococcus albus*. Those suffering from furunculosis are susceptible to that particular strain of staphylococcus with which they are infected.

Diagnosis is usually simple. Actinomycosis should be considered when incised and properly drained lesions do not heal.

Pathology In its early stages, a furuncle is hard and in-

durated. Either spontaneous involution occurs or the infected area softens in the center and a slough forms which is surrounded by a zone of tissue and vascular response (pyogenic membrane). To disturb this pyogenic membrane is to court disaster since it is the wall between tissues and the infection.

The pathology is characteristic. It consists of the formation of an abscess in the corium composed of a dense mass of polymorphonuclear leukocytes and lymphocytes. The lesion usually involves part of the hair follicle and its sebaceous gland.

Prognosis for solitary furuncles is good complete healing after incision and drainage occurs within a week. The prognosis in recurrent furunculosis in patients with lowered vitality is guarded. Several months may be required to overcome the condition.

Treatment. GENERAL MANAGEMENT This depends upon the stage in which the furuncles are seen. Sometimes they can be aborted in their early stages by applying a piece of adhesive plaster over the lesion. The "old-fashioned" poultice is mentioned only to condemn it as a means of spreading the infection. Any source of local irritation, such as tight or rough clothing chafing, etc. should receive attention. Uncleanliness about the body should be corrected by insisting on a daily hot soapy bath followed by alcohol applications. A general physical examination is usually necessary.

In the recurrent types all systemic and local factors should be remedied. A vacation with daily exposures to sunlight and attention to sources of focal infection often are rewarded with success.

SURGICAL. Suppuration can be hastened by applying hot packs. When "pointing" has taken place, and not before the furuncle should be incised, an iodoform drain inserted and a hot wet dressing applied. The surrounding parts should be sponged with a weak mercury bichloride solution or alcohol to inhibit any virulent bacteria in the vicinity.

Lip furuncles require special care. The patient should be hospitalized and hot compresses applied. Penicillin by injection (1 million units daily) with collateral x ray therapy is indicated in this type of case. Surgical measures are dangerous.

VACCINES are useful in the chronic recurrent cases. There is no advantage in the autogenous over the stock staphylococcus vaccine. Good results are possible from staphylococcus toxoid ambotoxoid or aerobacterin. When a staphylococcus ambotoxoid is used,

the injections are given at weekly intervals. The doses of less than 0.2 cc. are given intradermally (diluted with 0.3 cc. isotonic sodium chloride and the remainder subcutaneously in the deltoid area. The following scheme of dosage is used 0.05 cc. 0.1 cc. 0.15 cc., 0.2 cc., 0.25 cc., 0.3 cc. 0.4 cc. and 0.5 cc. In resistant cases the dosage may be increased to 1 cc.

PHYSIOTHERAPY X ray therapy is valuable in the treatment of facial lesions and axillary furunculosis. From 175 to 350 roentgen units of filtered radiation alleviate and shorten the course of the infection. General ultraviolet light therapy is useful in the disseminated cases, especially in children.

THE DIET should be light and low in carbohydrates. Indigestible foods, alcohol iodized salt, sea food and condiments should be excluded.

ANTIBIOTICS. While penicillin by injection is the drug of choice in acute furunculosis, if there is a history of penicillin sensitivity one of the other broad-spectrum antibiotics (Achromycin, preferably) should be given in doses of 250 mg. every 6 hours until the acute symptoms subside then the dose may be reduced. Topical antibiotics are not reliable in furunculosis as penetration is minimal.

SYCOSES VULGARIS

This pyogenic infection also called "barber's itch" is characterized by a deep invasion of the hair follicles. Early recognition is important as the disease is apt to become chronic if neglected. The condition starts as a red follicular papule which soon is converted into a yellow pustule pierced by a hair. The infected hairs are easily epilated because of the deep follicular involvement. The upper lip is the commonest site for the infection, although the entire bearded area may be involved. The axillary and the pubic areas the scalp and the eyebrows are involved infrequently. The intervening skin is usually inflammatory and painful. Permanent alopecia may result if the disease is not treated in its early stages.

Etiology The exciting cause is a strain of *Staphylococcus pyogenes aureus*. The disease is limited to males. Predisposing causes are shaving with a dull razor contamination with infected fingers or towels poor hygiene, nasal infection seborrhea and dusty or dirty occupations.

Pathology Syctosis vulgaris is a perifolliculitis with abscess formation

Differential Diagnosis

	<i>Sycosis Vulgaris</i>	<i>Tinea Barbat</i>	<i>Impetigo Contagiosa</i>	<i>Infections Eccematoid Dermatitis</i>
Course	Chronic	Chronic	Acute	Acute
Character- istic lesions	Pustules	Deep indurated nodules	Superficial crusted ulcers	Erythema and pustules
Bacteri- ology	Staphylo- coccus aureus	Tricho- phyton	Strepto- coccus	Staphylo- coccus or mixed
Distrib- ution	Limited to beard	Limited to beard	Lesions else- where	Upper lip

Prognosis. If the disease is treated early cure may result in from 3 to 6 weeks. The use of x ray therapy decreases the duration of the infection. Relapses are common in the undernourished alcoholics, those with focal infections, the unclean and in the cases with a seborrheic soil

Treatment. LOCAL THERAPY If the skin is very inflamed, hot saturated boric-acid or 1:5,000 mercury-bichloride or tyrothrycin packs are used for several days. Infected hairs should be epilated with forceps. This should be continued for several weeks after apparent cure. Chlorhydroxy-quinoline (Quinolone Ointment Squibb) starting with one fourth strength may be used at night. A trial of the various antibiotic ointments (except penicillin) may result in a good response. In my experience Aureomycin Ointment has proved useful. Relapses are common after cessation of treatment.

Autogenous vaccines and staphylococcus toxoid have not proved to be of value

X RAY THERAPY should be used if the disease does not respond to local treatment but x ray epilation is fraught with danger

FOLLOW UP Tonic measures and attention to focal infections are important. Shaving should be continued but the razor should be immersed in alcohol before using and the brush kept in an antiseptic solution. Seborrhea if present should be treated as well as nasal discharges otitis media, pyorrhea, blepharitis or sinusitis. An antibiotic cream should be applied to the entire area for several weeks after apparent cure to prevent relapse

PYOGENIC PARONYCHIA
(See p 542)

ACNE COMMOLOBATA

This disease also called dermatitis nodularis necrotica, consists of a wide-spread eruption of large indolent pustular acneform lesions usually occurring on the face, the back and the buttocks. In most cases the involvement is extensive with the eruption extending from the neck to the ankles. The lesions consist of large discrete indolent nodules, abscesses and sinuses.

Etiology These individuals have very large sebaceous glands. Once considered as occurring only in the tuberculous, present opinion regards the condition as not being caused by a specific organism but by various pyogenic "cocci." Biologically the condition is a special type of reaction occurring in a patient having a low state of resistance.

Treatment consists of lotio alba for local therapy and sulfonamide drugs, large doses of vitamin A, antibiotics stilbestrol and intravenous typhoid injections. In addition, debilitated cases improve from general exposures to ultraviolet light.

GRANULOMA PYOGENICUM
(See p 369)

PYODERMA VEGETANS

Pyoderma vegetans consists of reddish-brown papillomatous or verrucous masses due to the staphylococcus and occurs in dermatoses where moisture and poor hygiene predispose to secondary infection. The condition is not to be confused with pemphigus vegetans or blastomycosis which also are characterized by exuberant granulations.

Etiology Pyoderma vegetans may develop in patches of varicose dermatitis, tinea cruris and any moist dermatitis. It is more common in Negroes, the senile and the unclean. The patches, which are thickened, very vascular and often crusted, usually have well-defined borders.

Differential diagnosis is from blastomycosis and pemphigus vegetans.

Treatment. In the mild cases, wet dressings of potassium permanganate, boric acid or dalibour solution are helpful. In old cases x-ray therapy combined with foreign-protein injections has

been found to be of value. Destruction with the actual cautery is a method of last resort in the stubborn cases. Antibiotics locally or by injection also may prove to be useful.

FOLLICULITIS KELOIDALIS

Sycosis nuchae (acne keloid dermatitis papillaris capillitii) is a chronic perifolliculitis of the back of the neck, limited to men and characterized by a deep-seated infection of the hair follicles



FIG. 54 Pyoderma vegetans (Dr Harold Hunt)

with a tendency to keloidal scarring. In the early cases the lesions consist of pustules, papules and abscesses. After a variable period of time the lesions are replaced by fibrous nodules or a fibrous white or pink band. The disease is primarily a deep-seated staphylococcus infection.

Treatment. Better results are obtained if these cases are treated with x-ray therapy from the beginning. In extensive cases, fulgeration followed by x-ray therapy to prevent recurrence may be tried. Antibiotic ointments are of little value.

HIDRADENITIS SUPPURATIVA

(See p 535)

ACNE NECROTICA MILIARIS OF THE SCALP

This is a chronic persistent infection consisting of pinhead vesicopustules topped with small crusts, limited to the scalp and characterized by intolerable itching. Neurotic excoriations and an associated neurosis are often present. In some cases there is an oily seborrhea and a low threshold for pruritus.

Treatment consists of the application of $\frac{1}{4}$ strength chlorhydraxy-quinoline ointment or a 3 per cent ammoniated-mercury ointment at night and a 5 per cent alcohol solution of resorcinol monoacetate during the day. Office treatment consists of painting the lesions with a 2 per cent gentian-violet solution and graduated infections of staphylococcus toxoid. Ultraviolet light therapy to the scalp hastens resolution. If the itching is annoying, phenobarbital or a mild antihistamine should be prescribed.

FOLLICULITIS ABSCIDENS ET SUPPURIENS

This rare condition (multiple abscesses of the scalp) consists of individual and intercommunicating sterile linear and oval abscesses, absence of comedones, epidermal bridges, granulomatous ridges and sinus formation in the scalp followed by deep scarring. Usually the occiput is affected first. Although the boggy lesions are full of pus, the lesions are often sterile.

Etiology of the disease is unknown. Various pyogenic organisms have been found in the lesions. Most of the cases reported have been in Negroes.

Histologically it is an infective granuloma.

Treatment. Penicillin or tyrothrycin wet dressings and surgical drainage of the sinuses are indicated in the early cases but

are not always successful. The proper antibiotic drugs based on bacteriologic studies are important. Dermabrasion plastic surgery should be considered if scarring is extensive.

PYODERMA FACIALE

Pyoderma faciale is a rare pustular dermatitis affecting the face in young women and is characterized by resistance to treatment, a chronic course, deep destruction, ulceration, abscesses and sinuses.



FIG. 55. Pyoderma faciale. Note deep indurated, indolent abscesses and scars.

Etiology. Although the etiology generally is unknown, these patients usually are below par, underweight, and have moderate anemia, menstrual or endocrine dysfunctions.

Pathology. consists of localized necrosis involving the hair follicle and the adjacent sebaceous gland.

Treatment includes bed rest, tonics, a high-vitamin and high-caloric diet, daily antiseptic dressings of Zephiran 1:1000, correction of glandular dysfunction, autogenous vaccines or typhoid-vaccine fever therapy. The proper antibiotic drugs based on bacteriologic studies are important.

THE ACRODERMATOSES

These are various resistant pustular and eczematoid eruptions involving the hands and the fingers. Occasionally diagnosis is difficult without continued observation and bacteriologic aids.

ACRODERMATITIS CONTINUA (HALLOPEAU)

This low-grade infection, which is also called dermatitis repens, consists of pustules and eczematoid areas. The lesions first appear on the finger tips and nail folds and spread toward the palms or



FIG. 16 Dermatitis repens following trivial injury and characterized by an insistent weeping dermatitis, denuded epidermis and slow extension.

it may affect the soles. It is characterized by an undermining of the epidermis. Focal infection and a resistant strain of staphylococcus are etiologic factors.

This disease usually follows a banal injury. It is caused by the staphylococcus and in some cases *Ps. aeruginosa*. Biopsy shows superficial abscesses in the rete.

Prognosis is guarded because the disease is resistant to therapy and recurrences are common.

Treatment consists of débridement, potassium permanganate soaks and a trial of the broad spectrum antibiotics based on sensitivity tests. Focal infections should be removed, and the general health brought up to par.

PUSTULAR BACTERID

The pustular bacterid is a plaque consisting of sterile active or inactive vesicopustules caused by allergic dermal sensitivity to circulating bacteria or their products. They are found characteristically on the thenar and the hypothenar eminences of the palms and at the sides of the feet near the heels. There is no history or evidence of psoriasis.

Histologic section shows vesicopustules lying deep in the epidermis surrounded by little or no inflammation.

The diagnosis is made by finding a focus of infection; the presence of sterile pustules and improvement in a few cases following the removal of the focus of infection.

Prognosis is poor but the majority of patients eventually recover.

Treatment is unavailing. Antibiotics and the corticosteroids are not effective. The only beneficial effect results from the cautious use of a 3 per cent chrysarobin ointment. Cornia and Noun have reported success in some cases with quinacrine.

NURSING ASPECTS

Impetigo is the only member of the pyoderma group which is contagious and infectious on contact so that the nurse's hands should be protected with rubber gloves when treating a patient with this disease. Erysipelas is rarely infectious unless abrasions are present on the hands. The other members of the pyoderma group require a lowered state of resistance, bacterial sensitivity and focal infection for their development.

Impetigo in infants can be serious unless checked by standard procedures. Epidemics in nurseries often are started by the harboring of a "sore" on the skin by a member of the hospital personnel. If the mother develops impetigo, both mother and child should be isolated and sent home as soon as feasible. If more than one infant is infected the nursery should be vacated scrubbed and painted, the entire personnel should be examined for evidence of the disease and ointment jars and common articles used in the nursery should be examined for streptococci. The infants should be removed to private rooms and segregated into three groups, infected, exposed and new unexposed. The nurse should protect herself by the use of rubber gloves. She uses soap and water to remove the crusts, sterile scissors to snip the tops of the bullae and separate applicators for each application of the prescribed ointment. Since sulfonamide and penicillin preparations may induce sensitivity the safer antibiotics and silver nitrate solutions often are employed. Washcloths are taboo.

Erysipelas. Except in the aged and the debilitated as a result of serious accidents, this disease is no longer a problem since the advent of penicillin and other antibiotics. Sterile gloves should be used in applying dressings. The latter should be burned when soiled. The temperature and the pulse rate of febrile patients the progress of the spread of the disease and reactions to treatment should be noted.

Diseases Due to Vegetable Parasites

DERMATOMYCOSES

TRICHOPHYTOSIS CORPORIS

FAVUS

THE EPIDERMOPHYTOSES

TINEA CRURIS

RINGWORM OF HANDS AND
FEET

DERMATOPHYTIDS

POMPHOLYX

RECURRENT LYMPHANGITIS
OF THE EXTREMITIES

SAPROPHYTES

TINEA VERSICOLOR

ERYTHRASMA

OTOMYCOSES

THE BLASTOMYCOSES

BLASTOMYCOSES

COCCIDIOIDAL GRANULOMA

SPOROTRICHOSIS

DISEASES DUE TO RAY FUNGI

ACTINOMYCOSES

MYCETOMA

DISEASES DUE TO MONILIA

GENERALIZED CUTANEOUS

MONILLIASIS

MONILIAL VAGINITIS

FROSIO INTERDIGITALE

MYCOTIC PARONYCHIA

PFERLECHIE

MONILIDS

NURSING ASPECTS

THE superficial fungus diseases form a large percentage of the total number of cases seen in the practice of dermatology. In fact a considerable portion of the population is affected at one time or other by one or more of the common parasitic disorders. This state of affairs exists because the normal skin contains several species of saprophytic fungi, one or more dormant pathogens and numerous yeasts which may become activated under certain conditions.

The vegetable parasitic infections consist of diseases due to (1) the common superficial dermatotropic fungi which live in keratin material only (the skin, the hair and the nails) and do not invade the deeper tissues and (2) the rarer invasive fungi which often penetrate the deeper structures and become disseminated. These parasitic disorders range from a very mild superficial infection as tinea versicolor to the very serious infection, coccidioidal granuloma.

Fungus diseases have individual characteristics and must not be treated as a group reaction. Each type of fungus infection must be considered as a special therapeutic problem.

Cellular resistance to vegetable parasites is decreased by heat, moisture and friction which at the same time increase the permeability of the skin.

Infection with fungi under certain conditions may produce a generalized state of sensitization with the production of allergic or id lesions.

Diagnosis of fungus infections cannot be made accurately by inspection alone but must be aided by direct microscopic examination of the scales, the hairs or the tissues. In most cases, cultural studies are necessary also not only to determine the type of fungus but also for purposes of differential diagnosis.*

THE DERMATOMYCOSES

MICROSPOROSIS OF THE SCALP

(See p. 485)

TRICHOPHYTOSIS CORPORIS

This condition also called *tinea circinata* is an infection of the nonhairy parts with *Trichophyton* fungi resulting in single, multiple or disseminated lesions. The infection begins as a small red macule and enlarges by peripheral extension with central healing to form rings 0.5 to 4 cm. in size. The dry types tend to become scaly the moist types, vesicular or pustular. The rings may coalesce to form polycyclic lesions.

The eruption usually is localized at first on the exposed parts of the face and the neck later the trunk, the forearms and the legs may be involved. In rare cases infection may give rise to a deep granulomatous lesion *tinea profunda*.

Etiology The infection is usually acquired from stray cats, dogs or pets. Occasionally the eruption develops from a focus in the scalp. In some cases infection is acquired from some member in the family with scalp ringworm. Although the fungus is usually

*Consult the following books on mycology for additional information:
Leach, G. M. and Hopper, M. E. *Introduction to Medical Mycology* ed. 3 Chicago, V. Bk. Pub. 1948.
Simons, R. D. E. Ed. *Handbook of Tropical Dermatology* vol. II, New York, Elsevier 1953.
Simons, R. D. E. Ed. *Medical Mycology* New York, Elsevier 1954.
Swartz, J. H., and Rockwood, E. M. *Elements of Medical Mycology* ed. 1 New York, Grune & Stratton, 1949.

a *Trichophyton M lanosum M andomini* or *E. inguinale* may be found on cultures in other varieties.

Prognosis. Infections caused by *T violaceum* are difficult to cure while those produced by *M lanosum* respond to simple measures.

Pathology consists of an inflammatory reaction in the epidermis and the papillary layer with cellular infiltration and vascular dilation.

Differential Diagnosis. Impetigo contagiosa consists of a flat bulla covered with a yellowish crust, while the typical tinea lesion is covered with a thin moist scale with tiny vesicles at the periphery. New impetigo lesions may appear daily while tinea spreads more slowly. In seborrheic dermatitis there is less inflammatory reaction, scales are present, and vesicles are absent. The microscope is important in the differentiation.

The primary plaque of pityriasis rosea may be erroneously diagnosed as "ringworm." The generalized eruption which follows within a week confirms the examiner's suspicion.

Diagnosis is made by history, variation in size and inflammatory character of lesions, cultural studies, potassium hydroxide preparations or staining the scrapings with the Hotchkiss-McManus stain.

Treatment. Before applying medication the lesion should be scrubbed with tincture of green soap to remove the crusts and the cellular debris. For the superficial types the daily application of ✓ Vioform, Asterol, a mild sulfur, salicylic acid or undecylenic, propionic or $\frac{1}{2}$ strength Whitfield ointment is sufficient. For the deep types, a 10 per cent ammoniated mercury or a $\frac{1}{4}$ per cent Anthralin ointment in a water soluble base may be found to be necessary. The following ointment is useful.

✓ Precipitated sulfur	60
Salicylic acid	30
Water soluble base q.s.	1000
S Apply twice a day to the affected parts.	

FAVUS

Favus (*Tinea favosa*) is a chronic persistent fungus infection with the *Achorion schoenleinii* usually affecting the scalp but sometimes involving the trunk and the nails. The disease is contracted before puberty. It may remain dormant in the scalp for a long period causing a slow but progressive atrophic alopecia. The cutaneous lesions are similar to those found in the scalp and

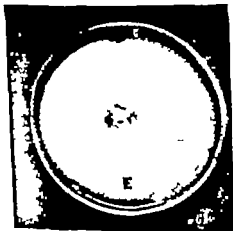
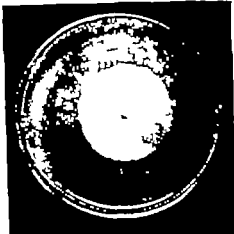


FIG. 57. Cultures of some common vegetable parasites. A *Achromon schenckii* B *Trichothyon sulphureum* C *Sporetrichum schenckii* D *Candida (Monilia) albicans* E *Macrosporum audouinii* (From Dr. Morris Moore)

consist of diffuse scaly areas or discrete, elevated round or oval yellow patches. These are covered with thick concretelike crusts which have depressed centers resembling a plate (scutula)

Etiology The disease is rare in the United States. Occasionally it is seen in recent immigrants or isolated rural residents. In some cases several members of a family are affected but epidemics are rare since the infectivity is low

Differential Diagnosis. *Tinea circinata* and monilial granuloma must be considered.

Treatment. Tincture of iodine or sulfur or ammoniated-mercury ointments are employed as local applications.



FIG. 58. *Tinea cruris*

THE EPIDERMOPHYTOSES

TINEA CRURIS

Tinea cruris (jock strap itch or red flap) is an acute subacute or chronic eruption which often affects the genitocrural region. The parts usually infected are the crotch and the inner third of the buttocks rarely the upper inner thighs the axillae the umbilicus and the submammary regions. Moderate itching is the rule

The acute types consist of single or multiple superficial patches of bright red dermatitis. Each area has a well-defined serpiginous border. The eruption may become eczematoid macerated or pustular from friction or poor hygiene

The chronic cases are more lichenified, usually bilateral and may spread downward along the inner thighs and around to the perianal region and the buttocks.

The color of the lesions varies from a light-brown shade in the inactive cases to a bright red color in the actively spreading patches. In the chronic cases there is more or less pigmentation. Maceration is common in the obese, the unclean and in those who perspire freely. Follicular or pityriasis rosealike "id" lesions may appear especially in overtreated or secondarily infected cases.

Etiology. Most cases occur in adolescent males. The condition is aggravated by sweat, uncleanness and friction. It is quiescent during the winter months and flares up in the summer. The disease usually is caused by the *E. inguinale* (floccosum). It is difficult to trace the origin of the disease, but locker rooms, gymnasium mats and toilet seats are a frequent source of infection. Those cases secondary to dermatophytosis of the feet can be traced to the bath towel.

Differential diagnosis is from seborrheic dermatitis, which is also found in the scalp and the axillary spaces and is more apt to occur during the colder months. Contact dermatitis, psoriasis, neurodermatitis and erythrasma must also be considered.

Prophylaxis. The patient should be stripped and the entire body examined for other sites of infection. The following precautions should be observed:

1. The patient should sleep alone, and the bed and the personal linen should be sterilized. Sexual intercourse should be avoided until the infection is controlled.
2. The bathroom and the toilet seat should be scrubbed with a 2 per cent cresol solution.
3. If the feet also are involved, proper therapy must be instituted at the same time to avoid reinfection.
4. The other members of the family should be examined for evidence of the disease.
5. The wearing of suspensories should be prohibited.
6. Exercise should be curtailed.

Prognosis is good but recurrences may occur from reinfection or discontinuing therapy too soon.

Treatment. ACUTE CASES with edema and maceration require bed rest and ice-cold packs of boric acid potassium permanganate (1:2,000) or Burrow's solution (1:15) in water or milk. After

the inflammation subsides and the disease enters the subacute stage the following lotion should be applied twice daily

Neocalamine	15.0
Zinc oxide	25.0
Lime water	15.0
Rose water q.s. ad	100.0

SUBACUTE CASES These are best treated by using 1 per cent resorcin in neocalamine lotion during the day and a propionic or undecylenic acid ointment at night.

CHRONIC CASES require more vigorous treatment. The following technic has proved to be 100 per cent successful. Paint the affected areas every 4 days with 3 per cent chrysarobin in carbon tetrachloride. Apply talcum powder as soon as the solution dries. The paint should be removed with soap and water after 8 hours. Any inflammation resulting can be controlled with calamine lotion. After the eruption clears, a fungistatic powder should be applied daily for several weeks.

RINGWORM OF THE FEET (TINEA PEDIS)

Ringworm of the feet (epidermophytosis or athlete's foot) is one of the common skin disorders.

Etiology Primary dermatophytosis of the hands is uncommon (see p. 625 for detailed discussion). The pedal type affects both sexes and all ages but is more common in adult males. Sweaty feet, poor hygiene, ill-fitting shoes, gymnasium showers and locker rooms are important factors in acquiring the disease. Most of the acute cases appear in the hot weather; chronic and sporadic cases occur at any season of the year. Maceration from any cause lowers the surface acidity of the skin and predisposes to infection. The common organisms are the *Trichophyton interdigitale*, *Epidermophyton inguinale* and *Monilia*. Fungi may remain dormant over a period of many months in calluses, nails or the skin itself. *T. mentagrophytes* usually is found in these lesions. *T. purpureum* infections are rebellious to treatment. Latent foci may flare up following injections of antibiotics.

Clinical Description. The clinical types which depend upon the strain of the fungus and the reactivity of the tissues are divided into three groups: (1) acute vesicular, (2) chronic intertriginous and (3) chronic squamous or hyperkeratotic. These may be complicated by secondary infection, fissures, nail involvement, eczematization, recurrent lymphangitis or dermato-

phytids. The fungi usually exist in a dormant state in the fourth interdigital space of the foot, which always should be examined routinely in all cases of ringworm of the hands and the feet. The plantar surface of the arch and the instep are also favorite sites. Itching is a frequent symptom but may be absent. Hyperhidrosis is usually present.

Complications include a superimposed contact dermatitis from irritating applications, dermatophytids (p. 188) secondary infection (vesico-pustules or cellulitis) eczematization or nail involvement.

Hot boracic packs rather than antibiotics should be used in those cases where secondary infection is present to avoid the danger of dermatophytids.



FIG. 39. Frused hyperkeratotic dermatophytosis.

Pathology The primary lesion is a vesicle. In the hyperkeratotic type the horny layers are thickened. Section shows intercellular edema, spongiosis and single or multiple intradermal vesicles. The infiltrate in the corium consists of polymorphonuclear leukocytes, while lymphocytes are often found about the vesicles.

Differential Diagnosis. Ringworm of the feet must be differentiated principally from contact dermatitis due to leather or dyes in the socks or the shoes. In these cases, the dermatitis is on the dorsal surfaces of the toes and the feet usually ending at the shoe tops. The soles and the interdigital spaces are not involved.

Diagnosis often is made on faulty and inadequate inspection and requires cultural or microscopic proof.

MICROSCOPIC See technic described on page 20.

CULTURAL. For differentiation the suspected material should be cultured on Sabouraud's maltose agar medium.*

Intradermal tests are of no practical value.

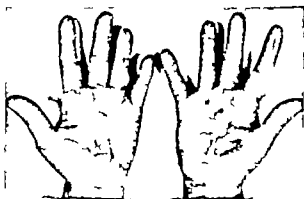


FIG. 60 Severe maceration of the palms resulting from over-treatment with Whitfield's ointment.

Prognosis. Minor uncomplicated cases usually clear up within a week following suitable therapy. Those cases with exzematization as a result of overtreatment, secondary infection, deep fissures, denuded areas or "id" manifestations may require several weeks of skillful therapy and close observation.

Treatment. There is no standard therapy for this disease since the management depends on the stage of the dermatitis, not on the possibility of a fungicidal onslaught against the invaders. Unfortunately, the treatment of this parasitic infection is not a simple matter for the following reasons: (1) localized or generalized allergy may exist (2) intolerance to drugs or ointments

*May be obtained from most laboratories or Derna Medical Co., Los Angeles 16, California.

may be present and (3) reinfection is common. The patient must be observed at frequent intervals in order to detect any untoward reactions to therapy.

✓ACUTE CASES are best treated with continuous wet dressings of 1:5,000 solutions of permanganate, saturated boric acid or Burrow's solution (1:15). All bullae should be opened, loose epidermis trimmed away and the entire area painted daily with 5 per cent potassium permanganate solution. Fungicidal ointments must never be used in this type of case as they will aggravate the eruption. The toes should be kept apart with lamb's wool. Bed rest is important to hasten involution and to prevent complications. As soon as the infection is under control, powders or lotions rather than ointments should be used to avoid the development of "id" lesions on the hands.

✓SUBACUTE CASES are treated with daily applications of one of the following: (1) 6 per cent benzoic acid and 6 per cent salicylic acid in alcohol (compound benzoic acid solution); (2) 2 to 4 per cent tincture of iodine; (3) 2 to 5 per cent silver nitrate solution; (4) stabilized Castellani's solution; (5) 2 per cent gentian violet in alcohol; (6) 1 per cent aqueous solution of permanganate; (7) 3 per cent Vioform, Timofax or Sopronol ointments are applied as a night dressing and removed in the morning. Local sensitization to these drugs may occur.

The macerated variety is best treated with 5 per cent silver nitrate in sweet spirits of niter for several days, followed by Lasar's paste.

CHRONIC CASES are best treated with ointments of tar, beta naphthol, Anthralin, sulfur, benzoic and salicylic acids, propionic acid or undecylenic acid. These preparations should be prescribed first in weak strengths to determine skin tolerance. Verdeflam (Texas Pharmacal Co.) may be useful in *T. rubrum* infections.

Whitfield's ointment (compound ointment of benzoic acid N.F.) is made up as follows:

Benzoic acid	12.0
Salicylic acid	6.0
Wool fat (anhydrous)	5.0
White petrolatum q.s.	100.0

If this ointment is used over too long a period of time applied too vigorously or used on sensitive skins, there is danger of maceration, edema and ulceration. (See Fig. 60.)

In cases characterized by thickening of the skin or extreme itching x-ray radiation by an experienced dermatologist is effective usually.

Prophylaxis is unsatisfactory because of the numerous possibilities of reinfection. The following methods are in use (1) sterilization of the bathtub the floors and footwear (2) foot powders containing boric acid, salicylic acid thymol sodium borate, hydroxyquinoline undecylenic acid, propionic acid (3) correct hyperhidrosis faulty footwear and hygiene (4) treat or remove foci of infection in nails or calluses and (5) apply 1 per cent tincture iodine to soles of feet and dorsum of toes after possible exposures. Patients with dermatophytosis should not receive penicillin therapy unless urgent, because of the great danger of "id" eruptions. If antibiotics are necessary it is safer to prescribe Mysteclin (Tetracycline plus Nystatin).

Mechanical scrubbing and flushing the floors and showers is preferable to chemical methods.

Criteria for Cure There must be complete cessation of itching and absence of scales and vesicles.

Personal Hygiene and Foot Care. The feet should be washed with soap and water at least once a day. After thorough drying (especially the interdigital spaces) any of the commercial foot powders should be used. Scuffs made of paper bath towel or wooden clogs should be used in the showers of public baths, swimming pools or hotels. A newspaper may be spread over the bath mat to avoid contamination from this source. Heavy shoes, rubber soles and tennis type shoes should be avoided in the summer months. Socks should be of cotton which may be boiled easily. Since infection may be acquired from leather linings, youngsters should be advised to wear anklets or socks when wearing shoes.

DERMATOPHYTIDS

Dermatophytids are allergic manifestations in sensitized individuals, resulting from absorption of fungi or their toxic products. Since the organisms cannot live in the corium cultural and microscopic examination for fungi are negative. The clinical varieties of these lesions include the following (1) eczematoid (2) pompholyx, (3) lichenoid (4) psoriasiform (5) crystalloid and (6) erythema nodosum. The type of clinical lesion produced depends on (1) the virulence of the fungus, (2) the depth of in-

fection, (3) the presence of epidermal dermal or vascular sensitivity (4) the degree of sensitivity

Etiology These lesions may be caused by (1) overtreatment, maceration trauma or secondary infection of the primary focus, (2) x-ray therapy (3) virulent fungi, (4) sensitized skin from previous infection and (5) penicillin injections.

Prognosis. These "ids" usually clear up within from 3 days to 3 weeks after control of the primary infection.

Diagnosis. The diagnosis of dermatophytids is made by the finding of a primary focus, a strongly positive trichophylin test and the absence of fungi in the lesions.

Treatment consists of drying lotions, control of the primary focus by bed rest, wet packs to promote drainage and proper care of infected areas. Acute generalized "id" eruptions often respond to Hydrocortone Prednisone or Prednisolone followed by gradual withdrawal. Antibiotics should be avoided. Dermatophytids that persist should receive courses of trichophyton injections (Hollister-Ster Laboratories)

POMPHOLYX

Pompholyx (dyshidrosis) is a general term used to describe deep vesicular eruptions on the palms and the soles.

Varieties (1) Functional, (2) mycotic, (3) contact and (4) toxic.

Clinical Symptoms. The eruption is usually symmetrical and bilateral, appears suddenly and is associated with more or less burning and tingling. The condition is limited to palmar surfaces. In some cases the eruption may spread to the dorsum of the hands and the fingers. Recurrent attacks are not uncommon.

The primary lesion is a deep-seated rounded vesicle with clear serous contents. Involution is by absorption followed by slight exfoliation.

Etiology THE FUNCTIONAL TYPE occurs in nervous patients and often are associated with hyperhidrosis.

THE CONTACT "ID" TYPE are caused by absorption of the products of inflammation from an area of exogenous dermatitis in a sensitized patient.

THE MYCOTIC TYPE. About 10 per cent of all cases of vesicular eruptions of the palms are "id" eruption resulting from allergy to a focus of epidermophytosis on the feet.

THE TOXIC TYPES. Some cases occur during acute febrile illness. Penicillin injections may be followed after 24 to 48 hours by an acute vesicular eruption of the hands and the fingers. The feet also may be affected.

Pathology There is no general agreement regarding the role of the sweat glands in this condition. These probably play an important part in the functional types of pompholyx.

Prognosis is poor in the functional types as recurrences are common unless there is a complete change in the mode of living or working.

Differential Diagnosis. In the functional types there is a history of frequent recurrences and appearance after mental fatigue or emotional excitement. In the contact "Id" types there is the presence of a contact dermatitis, patch tests are positive and the eruption disappears gradually with clearing of the primary dermatitis.

Treatment. In the functional types, x ray therapy and sedation are specific. The large vesicles which are often present in the allergic fungus types should be opened with a sterile needle and astringent wet packs should be applied. Astringent lotions e.g. calamine and zinc oxide or Burow's solution (1:10) are also beneficial. The allergic mycotic types often respond to "peeling" therapy for which one half strength Whitfield ointment is applied for a few days. If a liquid is preferred the following application may be used 3 times daily.

Salicylic	oid	6
Benzoic	oid	12
Glycerine		5
Iodoform alcohol q.s. ad		100

(Discontinue as soon as desquamation occurs and follow with a cream e.g. Nivea.)

RINGWORM OF THE NAILS (See Onychomycosis p. 543)

SAPROPHYTES

TINEA VERSICOLOR

Tinea versicolor (liver spots) is a superficial eruption of light brown macules on the chest and the trunk, usually occurring in the summer months. It is caused by the *Malassezia furfur*.

Clinical Description. The eruption consists of circumscribed superficial round or irregular patches sometimes arranged in a

reticulated pattern. The absence of inflammation is a characteristic symptom. The lesions are yellowish reddish brown or dark brown and are covered with fine branny scales which contain myriads of fungi and spores. The upper third of the chest, the back and the shoulders are involved, although the submammary regions, the anterior axillary spaces and the genitocrural creases may be affected also.

The disease usually becomes more noticeable following exposure of the body to the sun. In patients thus exposed a reticulated pseudo-achromia may develop from filtration of light by the fungi. As a rule there are no symptoms. Slight itching is present in the acute cases.

Etiology While the disease is commonly believed by the laity to be caused by a liver disturbance the causative factor is a local infection with *Malessezia furfur* in susceptible individuals. Apparently a tendency to sweat profusely, a favorable soil and prolonged contact are necessary for infection. This common condition usually is found in the 20 to 40 age group.

Differential diagnosis is from vitiligo and postinflammatory pigmentation.

Diagnosis is made on distribution, superficial character of lesions, history of seasonal recurrences, presence of fungi in the scales. Collect the scales on a clean slide after scraping with a dull sterile knife and add a few drops of 30 per cent solution of potassium hydroxide. After 1 hour examine under the high power (dry) objective for the typical spores and hyphae.

✓Treatment

1 The patient first takes a hot bath, using yellow laundry soap to remove the scales.

2 A saturated solution of sodium thiosulfate is then applied vigorously every night to the entire region. This should be followed by a hot sulphur soap bath in the morning.

3 If the eruption does not disappear within 10 days the following ointment is indicated.

Precipitated sulfur	50
Salicylic acid	10
Water absorbent base	qs 100.0

4 Treat the disease for at least two weeks after it apparently has disappeared to avoid recurrences.

5 All bed linen and underclothing should be laundered to prevent recurrence

6 Ultraviolet light and sunburn on the affected parts may result in pseudodepigmentation.

ERYTHRASMA

Erythrasma is a rare superficial fungus infection usually involving the axillary spaces and the thighs where two exposed surfaces rub together. The condition is similar to tinea cruris but is characterized by slower evolution, lack of elevation, absence of itching, dark brown color and minimal inflammation.

In chronic cases the lesions are slightly infiltrated or lichenified but never vesicular. When the disease is at its height, it acquires a reddish or a brownish color. The condition is caused by an infection with *Actinomyces minutissimus* which consists of very fine mycelia and small spores. There is some difficulty in recognizing these fungi in ordinary potassium hydroxide preparations unless the oil-immersion lens is used.

The treatment is similar to that of tinea versicolor.

OTOMYCOSIS

Fungus infections of the ears is a term employed frequently but erroneously for dermatoses of the ears and ear canals. Cases do occur in tropical climates but are not common. If fungi are found in cultures from the involved area, they are usually saprophytes or contaminants. Eczematoid dermatitis of the ears is frequently seborrheic dermatitis.

Treatment. True otomycosis of the ear canals usually responds to applications of Cressatin, Aerosporin otic sol. (B W) or Otobiotic sol. (White Lab). Seborrheic and infectious eczematoid dermatitis of the ears are treated with Vioform antibiotic or Neocortef ointments.

THE BLASTOMYCOSSES

BLASTOMYCOSIS

Blastomycosis (Gilchrist's disease) is a chronic infection localized or systemic caused by *Blastomyces* (double-contoured yeasts) and characterized by the formation of granulomatous lesions. The localized types consist of large ulcers covered with thick crusts or papillomatous vegetations. When the disease has

persisted for some time, numerous discharging sinuses are present. In some cases multiple lesions may result from autoinoculation. The lesions have a violaceous hue. The center of the patch is depressed with some atrophy while the characteristic border is elevated, verrucous and contains numerous millary abscesses which exude pus. The disease usually occurs on the exposed parts, including the dorsum of the hands the neck and the face.

The systemic type is characterized by progressive wasting, pyrexia lymphadenopathy toxemia and symptoms pointing to the involved organs. Superficial and deep nodules crusted ulcerations or verrucous draining sinuses, or furunculoid lesions appear on the skin. In this type of blastomycosis a roentgenogram of the chest should be made to rule out pulmonary involvement (90 per cent). The bones and urogenital organs not infrequently are involved.



FIG. 61 Blastomycosis. This type must be differentiated from bromide eruptions. (From Dr. R. O. Noojns)

Etiology Predisposing cause in many of the cases is a traumatic break in the skin. The causative yeast exists as a saprophyte on various plants. Adults usually are affected. Most of the cases in the United States have been reported from Chicago and vicinity although sporadic cases occur elsewhere.

Pathology Section shows microscopic abscesses and sinuses. The epidermis contains characteristic verrucous changes and a moderate or a marked acanthosis or pseudo-epitheliomatous hypertrophy. *Blastomyces dermatitidis* usually are found in the corium as double-contoured yeasts. A nonspecific cellular infiltration and a few giant cells are also present.

Diagnosis. In all cases a biopsy should be made and the section should be searched for yeasts and stained with the McManus stain. Repeated smears from the pus as well as cultures, intra scrotal injection of suspected material in mice should be made in doubtful cases. The urine should be cultured to detect early involvement of the genito-urinary tract. An intradermal test using blastomycin has been useful in the systemic types (may be negative in early and terminal cases).

Differential Diagnosis is from tuberculosis verrucosa cutis, granuloma inguinale, leoderma or bromoderma.

Treatment. X-RAY THERAPY when given with large doses of potassium iodide (200 grains daily) is effective in the uncomplicated cases. When the viscera are involved the cure depends upon the extent of the involvement, the presence of allergy and the damage to the vital organs. Snapper and McFar Jr (Am. J. Med. 15:603, 1953) report excellent results in the systemic types with Hydroxystilbamidine Isethionate (Merrell). The drug is administered in graduated doses from 50 to 150 mg. in 150 cc. of 5 per cent glucose in normal saline. Injections are given by slow intravenous drip daily or every other day for several weeks until the patient is afebrile, the lesions healed and the cultures negative. Circulatory collapse and hepatotoxic and neurotoxic effects are side reactions.

Prognosis. The local types respond well to x ray and iodide therapy although recurrences are frequent in incompletely treated cases. Visceral involvement is always a serious complication.

COCCIDIOIDAL GRANULOMA

Coccidioidal granuloma (California disease) is a chronic infectious disease caused by the *Coccidioides immitis* and is char

acterized by skin lymph-node bone and visceral involvement.

Clinical Symptoms. In the *local* types the lesion is similar to that of verrucous tuberculosis. The *systemic* cases are characterized by crops of painless subcutaneous abscesses or infiltrated granulomatous nodules. The early symptoms, which are those of an upper respiratory or influenza infection (valley fever) often are unrecognized. In this stage there is a characteristic eruption of erythema nodosum. The late lesions consist of involvement of the bone, meningitis and bronchopneumonia. Blood cultures are rarely positive. If the organisms gain entry into the body by inhalation of the spores bronchopneumonia or blungland involvement may develop. The systemic types with metastatic skin lesions and inanition often are confused with blastomycosis. The incubation period varies from 7 to 28 days.

Types. (1) The primary benign or localized form. (2) The rare progressive systemic or disseminated type.

Etiology The specific cause is the *Coccidioides immitis* which is a spherical highly refractive, double-contoured yeast. Reproduction is not by budding but by endogenous spore formation. It produces a cottonlike growth on culture media. Over 90 per cent of the cases occur in central and southern California. Males, especially agricultural workers between the ages of 20 and 25 and Negroes and Filipinos are predisposed. The spores enter the skin through abrasions or by way of the gastro-intestinal tract or via inhalation of contaminated dust.

Differential Diagnosis. The *bronchopneumonic* type may be mistaken for miliary tuberculosis but the sputum is negative for tubercle bacilli. Blastomycosis and chronic glanders must be ruled out by cultural studies.

Diagnosis usually is made by presence of organisms in the sections, hanging-drop preparations from the sputum or the pus or by intraperitoneal injections in male guinea pigs and a special complement fixation test. The value of the intradermal coccidioidin test is somewhat uncertain in the disseminated types.

Prognosis. While spontaneous remissions occasionally occur the disseminated types are usually fatal. The early pulmonary cases may get well.

Treatment is mainly supportive as no specific therapy is available at this time. Ethyl Vanillate (Squibb) and Progesterone are being used in the systemic cases, but results are inconclusive.

SPOROTRICHOSIS

Sporotrichosis is a chronic infectious disease characterized by chainlike nodules and is caused by the *Sporotrichum schenckii* and other varieties. The disease consists of a nodule pustule or verrucous plaque (sporothrix chancre) which develops at the portal of entry and an ascending regional lymphangitis and multiple subcutaneous indolent, painless abscesses and ulcers. In rare instances the disease becomes systemic with the production of visceral and bone lesions as well as disseminated cutaneous abscesses.

Etiology The disease is due to the sporothrix which exists as a saprophyte on plants and brush salt hay peat moss or on flower bulbs. Gardeners, farmers and laborers are predisposed. The organism is easily cultured on Sabouraud's or Littman's agar producing white colonies with radiating filaments. It is difficult to find the organisms in ordinary smears.



FIG. 62 Culture mount of *Sporotrichum schenckii* showing typical spores attached to short stalks and mycelia. (Dr. D. C. Smith's case)

Clinical types include (1) lymphatic, (2) disseminated nodular (3) disseminated ulcerative and (4) visceral. In this country the lymphangitic type is most prevalent.

Diagnosis. The history, the occupation, the clinical picture of ascending lymphangitis with indolent abscesses, the positive sporotrichin test, positive animal inoculation (mice) tests, cultural studies and rapid involution of the lesions following suitable iodide therapy help in making the diagnosis. It is difficult to demonstrate the spores in wet smears.

Differential diagnosis is from furunculosis, tularemia, late syphilis and primary tuberculosis complex.



FIG. 63 Sporotrichosis. The primary lesion is on the finger. The infection travels along the lymphatics, leaving chain of nodulo-ulcerative lesions (From Dr D. C. Smith)

Pathology A study of the section shows the lesion to be an infectious granuloma. The organism which is easily cultured, is difficult to find in sections or pus. Pear shaped or cigarlike spores may be demonstrated in the smears from cultures.

Prognosis is good in the cutaneous types, serious in the visceral forms.

Treatment. Potassium iodide therapy (saturated solution) con-

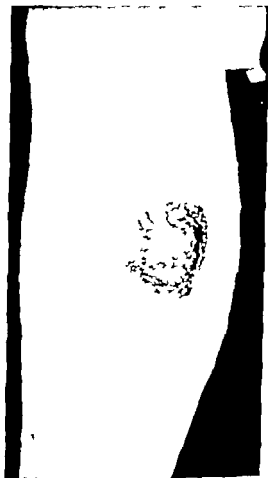


FIG. 64 Primary actinomycosis of the skin. Source of infection is unknown.

sisting of from 30 to 100 gr daily is usually sufficient to cure the average case. The treatment should be continued for about a month after apparent cure to prevent relapse. If the lesions are open, they should be dressed with a 5 per cent sodium-iodide ointment.

DISEASES DUE TO RAY FUNGI

ACTINOMYCOSIS

Actinomycosis ("lumpy jaw") is a chronic infectious disease due to actinomyces infection.

Clinical Symptoms The primary lesion may occur in the skin, the lungs or the gastro-intestinal tract. The cutaneous type starts as a small indolent pustule which persists after incision and enlarges gradually. In the cervicofacial type the organisms enter the deep tissues through carious teeth or periodontal pockets. The onset is insidious with pain in the region of the jaw gradual edema and infiltration. The neck becomes stiff and eating becomes more difficult. After a variable length of time the lymph nodes become deep-seated boggy swellings. These later develop into livid red tumors with sinus formation. Upon pressure a bloody or purulent fluid containing sulfur yellow granules, which consist of masses of fungi, escapes from the sinuses.

In the vertebral type the spine becomes involved secondary to the skin involvement. The abdominal type is characterized by the presence of numerous abscesses on the abdominal wall.

Differential Diagnosis is chiefly from nocardiosis, squamous-cell carcinoma, blastomycosis, and pseudoepitheliomatous hyperplasia.

Etiology The organisms probably exist in the mouth or in carious teeth as a saprophyte but becomes pathogenic as a result of trauma (tooth extraction) and unknown factors. The theory that the disease is acquired by contact with hay straw or vegetable matter harboring the saprophytes has not been established.

Pathology Section shows numerous nodular masses of granulation tissue containing fungus granules with radiating filaments which terminate in clublike nodes. The infiltrate surrounding the masses is characteristic of an infective granuloma.

Prognosis is best in the skin and the cervicofacial types, poor in the thoracic and the abdominal types. The disease is serious because of the danger of visceral involvement. Death may occur from septicemia, lung or brain abscess, peritonitis or meningitis.

Treatment. In the cutaneous types without visceral involvement, massive iodide and x ray therapy are indicated. For the visceral types, surgical drainage and ten million units of penicillin or one of the following antibiotics Aureomycin Terramycin or Chloromycetin. Strains of the organism vary in their response. We prefer Aureomycin in the following dosage 750 mg every 6 hours for 10 days then 500 mg every 6 hours for 10 to 21 days or longer. The sulfonamide drugs may be effective in antibiotic resistant cases.

MYCETOMA

Mycetoma (Madura foot) is a chronic fungus disease of the skin caused by various types of fungi Imperfecti including *Madurella* and is characterized by deep subcutaneous abscesses of the foot which produce swelling and distortion of the foot.

Clinical Symptoms. The foot gradually becomes swollen and boggy with nodule and sinus formation. Examination of the secretion from the sinuses reveals numerous granules of gray red or black fungi resembling fish roe. As a result of the chronic invasive infiltration a characteristic marked deformity of the foot results. In the neglected cases osteitis and osteomyelitis occur as complications.

Etiology The organisms enter the skin through a minor injury which may be unnoticed by the patient. In the United States the disease is practically limited to barefooted Negroes. Over 15 varieties of fungi have been reported to have been found in the lesions.

Differential diagnosis is from late syphilis and tuberculosis.

Treatment. Massive iodide therapy and antibiotics may be of service. If the subcutaneous tissues are soft and boggy they should be removed by curettage. In the extensive cases with bone involvement amputation is the only means of curing the disease.

DISEASES DUE TO MONILIA

Monilliasis includes a large group of infections of the skin and the mucous membranes caused by yeastlike fungi belonging to the *Monilia* (*Oridium*) group of organisms. Systemic infections (pulmonary intestinal etc.) are rare but often fatal. Of the half a dozen *Monilia* found on the skin and the mucous membranes *Candida albicans* is the only true pathogen but the ubiquity of the



FIG. 65. Monilliasis of the skin. Generalized cutaneous type of lesions with systemic involvement. Note the parulis at the angles of the mouth and the typical pustular and crusting lesions of the forehead and the chest (From Dr. Wesley W. Wilson)

organism requires extreme caution in evaluating laboratory diagnosis. *C. albicans* grows readily on corn meal blood, dextrose or Sabouraud's agar with the production of creamy colonies having a yeasty odor. Budding cells with or without filaments can be recognized.

Monilial infections may be divided into (1) general cutaneous infections, (2) systemic infections (pulmonary, gastro-intestinal, endocardial and meningitic), (3) local infections and (4) monillids.

GENERALIZED CUTANEOUS MONILLIASIS

Cutaneous monilliasis is a parasitic infection of the skin and the mucous membranes, caused by *C. albicans* and is characterized by a polymorphous eruption involving the flexure surfaces and regions of the body which usually are irritated by friction. The infection tends to involve the inframammary and the anogenital regions, the buttocks, the axillae and the flexures of the elbows

Treatment. In the cutaneous types without visceral involvement, massive iodide and x-ray therapy are indicated. For the visceral types, surgical drainage and ten million units of penicillin or one of the following antibiotics Aureomycin, Terramycin or Chloromycetin. Strains of the organism vary in their response. We prefer Aureomycin in the following dosage 750 mg every 6 hours for 10 days then 500 mg every 6 hours for 10 to 21 days or longer. The sulfonamide drugs may be effective in antibiotic resistant cases.

MYCETOMA

Mycetoma (Madura foot) is a chronic fungus disease of the skin caused by various types of fungi imperfecti including *Madurella* and is characterized by deep subcutaneous abscesses of the foot which produce swelling and distortion of the foot.

Clinical Symptoms. The foot gradually becomes swollen and boggy with nodule and sinus formation. Examination of the secretion from the sinuses reveals numerous granules of gray red or black fungi resembling fish roe. As a result of the chronic invasive infiltration a characteristic marked deformity of the foot results. In the neglected cases osteitis and osteomyelitis occur as complications.

Etiology. The organisms enter the skin through a minor injury which may be unnoticed by the patient. In the United States the disease is practically limited to barefooted Negroes. Over 15 varieties of fungi have been reported to have been found in the lesions.

Differential diagnosis is from late syphilis and tuberculosis.

Treatment. Massive iodide therapy and antibiotics may be of service. If the subcutaneous tissues are soft and boggy they should be removed by curettage. In the extensive cases with bone involvement amputation is the only means of curing the disease.

DISEASES DUE TO MONILIA

Monilliasis includes a large group of infections of the skin and the mucous membranes caused by yeastlike fungi belonging to the *Monilia* (*Ordnium*) group or organisms. Systemic infections (pulmonary, intestinal etc.) are rare but often fatal. Of the half a dozen *Monilia* found on the skin and the mucous membranes, *Candida albicans* is the only true pathogen but the ubiquity of the



FIG. 65 Moniliasis of the skin. Generalized cutaneous type of lesions with systemic involvement. Note the perleche at the angles of the mouth and the typical pustular and crusting lesions of the forehead and the chest (From Dr Wesley W. Wilson)

organism requires extreme caution in evaluating laboratory diagnosis. *C. albicans* grows readily on corn meal, blood dextrose or Sabouraud's agar with the production of creamy colonies having a yeasty odor. Budding cells with or without filaments can be recognized.

Monilial infections may be divided into (1) general cutaneous infections, (2) systemic infections (pulmonary gastro-intestinal endocardial and meningitic) (3) local infections and (4) monilids.

GENERALIZED CUTANEOUS MONILIASIS

Cutaneous moniliasis is a parasitic infection of the skin and the mucous membranes, caused by *C. albicans* and is characterized by a polymorphous eruption involving the flexure surfaces and regions of the body which usually are irritated by friction. The infection tends to involve the inframammary and the anogenital regions the buttocks the axillae and the flexures of the elbows

and the knees the lips (perlèche) the mouth, the tongue and the nails. In most of the cases the eruption is papulosquamous or papulovesicular. When the lesions coalesce, they form patches with scalloped edges.

Etiology Predisposing factors include anemia, dehydration, hyperglycemia, hypovitaminosis B-complex and obesity. Most of the fatal cases occur in children.

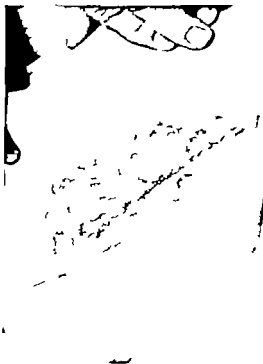


FIG. 66. *M. nifasus* of the breast in an obese patient. Glucose tolerance test revealed mild diabetes.

Diagnosis Generalization of the characteristic lesions, repeated positive cultures of *C. albicans*, positive stool cultures, positive agglutination tests and gradual emaciation are diagnostic.

Treatment

Generalized *C. albicans* infections are resistant to therapeutic measures. There appears to be a systemic anergy in these cases.

which occasionally can be corrected by reduction diets in the obese polyvitamins, fever therapy or Diiodoquin.

Nystatin (Mycostatin Squibb) has improved the prognosis in these cases. The dose is one 500,000 unit tablet T.I.D. until the stools are negative and the skin and mucous membrane lesions have cleared.

THRUSH

(See Chronic Oral Monilliasis p. 561)

MONILIAL VAGINITIS

Monilial vaginitis is caused by infection with *Candida albicans*. The clinical picture consists of a thick caseous material on the vaginal walls, superficial reddened denuded areas with scalloped edges on the vulva and numerous excoriations from scratching. Pruritus and soreness aggravated by urination are frequent complaints.

Etiology The organisms are found in about 10 per cent of normal vaginal tracts. Infection sometimes results from monilial balanitis in the husband. Pregnancy malnutrition diabetes and vitamin deficiencies are predisposing causes. Pregnant women with monilial vaginitis may infect the fetus as it passes through the birth canal.

Treatment consists of daily applications of one of the following: (1) a 1 to 5 per cent aqueous solution of gentian violet, (2) vaginal suppositories of Mycostatin (3) propionate vaginal jelly and weak white vinegar douches. The systemic cause must be investigated and treated to avoid recurrences.

EROSIO INTERDIGITALE

This is a common condition that involves the interdigital spaces of the fingers. It is characterized by the presence of a moist, white sodden epidermis. In most of the cases the webs of the fingers of the third and the fourth interspaces are affected. When the sodden epidermis is removed a glistening red base is exposed which is tender to the touch. The condition is fairly common in obese housewives, latent diabetics, dishwashers, fruit canners and others who have their hands in contact with strong alkali solutions.

Treatment. If protective measures are not instituted the disease will be found to be resistant to treatment. The application

of 2 per cent alcoholic gentian violet solution 5 per cent silver nitrate Mycostatin ointment or $\frac{1}{10}$ to $\frac{1}{4}$ per cent Anthralin in Lassar's paste is usually effective.

To avoid recurrences, one of the plastic resins (e.g., Covitone) should be used if the hands are continually immersed.



FIG. 67 Extensive monilia in a bed fast patient

MYCOTIC PARONYCHIA

This is an acute or chronic bolsterlike inflammation of the nail fold usually multiple caused by *C. albicans*. The nails of the affected fingers assume a pitted fissured and pigmented appearance. The condition occurs in housewives bartenders, soda fountain workers and employees of canneries who come in contact with sugar solutions. *C. albicans* is usually found in the scrapings.

Diagnosis is made by the history the occupation the tendency of multiple involvement and the staining of scrapings with the Hotchkiss-McManus stain.

Treatment consists of avoidance of prolonged contact with water and the local application of a 2 per cent alcoholic solu-

tion of gentian violet under the nailfolds and dressings of Vioform cream or Mycostatin ointment.

PERLECHE

Perleche is a macerated, eroded or fissured condition of the corners of the mouth. Although the juvenile types are usually due to the *Streptococcus*, the adult cases are frequently caused by *C. albicans* lip-licking or macroglossia. Some of the cases in elderly individuals result from maceration caused by the excessive salivation from poorly fitting dental plates. In some instances the vulvar labiae or the anal orifice may be involved. A similar con-

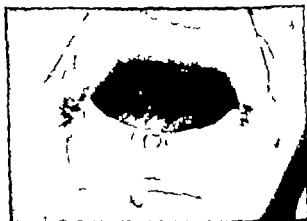


FIG. 68 Perleche *Candida albicans* infection in a diabetic patient.

dition may occur at the corners of the eyelids from using astringent eye drops.

Treatment consists of applications of compound benzoin tincture and Mycostatin ointment.

MONILIDS

Monillids are allergic polymorphous eruptions which develop in an individual whose skin is sensitized by a primary focus. The eruption is follicular, papular or erythematous-squamous. The diagnosis is made by the presence of a primary focus and a strongly positive oidiomycin reaction.

Treatment consists of therapy directed to the original focus.

NURSING ASPECTS

The "ringworm" group of dermatoses are not infectious if ordinary aseptic precautions are observed. The physician will desire culture media, sterile forceps, small sterile scissors a plating rod and 20 per cent solution of potassium hydroxide. Paper slippers should be provided for patients with "athlete's foot." Hospital patients should be advised to use wooden clogs when using the bathroom facilities to avoid contamination of the showers or the floors. The nurse should be aware of the fact that the use of parenteral antibiotics may cause a flare-up of latent foci of ringworm infection (feet, anogenital areas and groins)

Blastomycosis, actinomycosis and coccidioidal granuloma are rare serious types of yeast infections. When limited to the skin these usually are curable but when they are disseminated through the blood stream internal organs often are involved, often with fatal consequences. Rubber gloves should be worn when bathing or dressing these cases, and soiled dressings should be burned.

Monilliasis usually responds to local therapy. Blood sugar studies usually are made to determine the presence of diabetes, and stools are examined for the presence of the organisms.

The nurse should avoid the careless use of the word "ringworm" in treating hand cases since fungus infections of the hands are uncommon. Many dermatoses which occur in rings are treated injudiciously for ringworm resulting in serious inconvenience to the patient.

Diseases Due to Animal Parasites

ECTOPARASITES

SCABIES
 PEDICULOSIS CORPORIS
 PEDICULOSIS PUBIS
 FLEASITES
 RAT-MITE DERMATITIS
 BEDBUG BITES
 TICKS
 CHIGGERS (RED BUGS)
 DEMODEX FOLLICULORUM

ECTOPARASITES (Contd.)

MOSQUITOES, HORSEFLIES
 AND GNATS
 MISCELLANEOUS PARASITES
 CREEPING ERUPTION
 SCHISTOSOME DERMATITIS
 (SWIMMER'S ITCH)
 ENDOPARASITES
 RAT BITE FEVER
 NURSING ASPECTS

General. A broad classification of animal parasites includes (1) the ectoparasites, which involve the epidermal structures only e.g. scabies and pediculosis and (2) the endoparasites which involve the blood stream and the viscera, e.g., amebiasis cutis and rat bite fever.

Skin lesions caused by biting insects may result from mechanical trauma secondary infection injection of toxic substances and injection of a normally harmless substance into a previously sensitized skin. Contact dermatitis at the site of the bite is frequently seen from the application of irritating antiseptics or antipruritic ointments.

This group of dermatoses may cause much discomfort may become an economic or military problem and may result in disabling illness.*

ECTOPARASITES

SCABIES

Scabies (seven years itch) is a contagious disease characterized by nocturnal itching red follicular papules, burrows and evidence

*For a detailed discussion of parasites and the parasitic eruptions in the human, see Craig, C. F. and Faust, E. C., *Clinical Parasitology*, ed. 2 Philadelphia, Lea & Febiger 1943 Also Mitchell Heggs, G. B. ed. *Modern Practice in Dermatology* chaps 22-23, New York, Hoeber, 1950.

type is more readily cured than the human variety since these parasites do not live long on the human skin. The eruption consists of a red papular or urticarial rash which is limited to the forearms, the elbows, the neck and the face. Fowl mites are small red parasites. The affected animal should be destroyed to prevent further infection.

Differential Diagnosis. Scabies must be differentiated in children from papular urticaria, in which there is both diurnal and

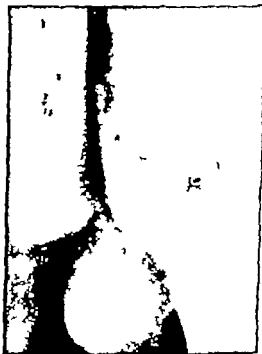


FIG. 71 Typical involvement of buttocks in scabies

nocturnal itching, face involvement, central necrosis in the lesions, dermographism and the absence of burrows. Pediculosis corporis affects the shoulders, the interscapular region, the waistline and the upper half of the buttocks. Neurotic excoriations in the institutional psychotic may cause confusion. In these patients the hands and the genitals (males) are not involved, scars from healed



FIG. 72. Involvement of the hands in scabies.

excoriations are present and a therapeutic test with benzyl benzoate does not affect the pruritus or the lesions.

The generalized pruritus with excoriations, which may occur in diabetes and lymphoblastomas, does not affect the typical scabetic sites, and the pruritus is cyclic rather than nocturnal. Vesicular eczematoid eruptions on the hands with disseminated "Id" lesions and early cases of dermatitis herpetiformis sometimes may simulate scabies.

Diagnosis. The patient should be stripped and the sites of involvement noted. There are no typical lesions, only scratch marks, a few impetiginous lesions in children and possibly a few vesicles on the lateral surfaces on the hands, on the wrists or between the fingers. The diagnosis should be withheld from the patient until the examiner is sure of his diagnosis. It is not necessary to find the mite or the burrow in order to make a diagnosis of scabies. For demonstration purposes, however the mite can be removed from the burrow by inserting a needle into the open end of the tunnel parallel with the surface. The mite will be found at the blind end clinging to the needle point. It can be mounted in glycerin and examined under the low-power lens of the microscope.

If no burrow can be found scrapings from under the nails may contain parts of the parasites. The presence of generalized itching at night may be the only symptom in individuals who bathe frequently. The face and the scalp are never involved in adults. The presence of other cases in the family and the involvement of one or more characteristic sites should aid in the diagnosis. A therapeutic test also may be helpful.

When clinical evidence of the disease is atypical a papule or an excoriation may be scraped, and the scrapings transferred to a glass slide. A drop or two of glycerin is added and, under a low power search is made for acari ova, eggshell moults or scybala.

Treatment. All members of the family are treated at the same time regardless of the presence or absence of itching or lesions. Bedclothes, blankets, socks and underwear are removed first and laundered. The outer clothing should be sent to the cleaners. Leather wrist watch bands also should be cleaned. The cleaning of hats and outer clothing will prevent acarophobia on the part of the patient. If secondary infection is present the scabies should be treated first. The choice of the preparation used depends on the availability of the product, the fastidiousness, the financial resources and the co-operation of the patient and the results of previous therapy, if any.

BENZYL BENZOATE EMULSION (25 per cent) is easy to apply and effective in 90 per cent of the cases. The patient takes a hot bath thoroughly scrubbing the affected parts. The emulsion is applied carefully from the neck down with a brush or a cotton swab. After 24 hours the emulsion is reapplied but it is not advisable for the patient to take a bath before the second application. Then treatment should be discontinued for at least a week to determine the effect of the drug and to avoid over treatment.

KWELL OINTMENT (gamma benzene hexachloride) is applied in the same manner as Benzyl benzoate one application often being sufficient. If itching persists, repeat in 5 days.

EURAX (crotonotoluide) is effective in many cases and requires two applications 24 hours apart.

GAMFEGENT OINTMENT (National Drug Co.) a combination of pesticides usually is effective within 24 hours after one application.

CIDALON (Canada Pharmacal Co.) which contains 4 per cent isobornyl thiocyanacetate is effective also.

TOPOCINE (Lilly) is a combination of benzyl benzoate D.D.T and benzocaine.

Any increase in itching and the presence of signs of dermatitis within 24 hours after the application of any of the above preparations indicates sensitization, and prompt withdrawal of the medication is imperative. Soothing lotions are necessary for several days until the dermatitis clears up after which the entire skin should be studied for evidence of persistence of the infection. If nocturnal itching and fresh papulovesicles continue to appear in the usual scabietic sites treatment should be resumed with another type of antiscabietic, and not the one which caused the contact dermatitis.

The principle of therapy is to avoid overtreatment as well as undertreatment. The choice of drug used is not as important as the thoroughness with which it is employed.

PEDICULOSIS CAPITIS

(See p 492)

PEDICULOSIS CORPORIS

This is characterized by a pruritic eruption on the trunk which consists of scratch marks and pigmented lesions. It is caused by an infestation of the clothing with body lice (*Pediculus vestimentii*). The itching is both diurnal and nocturnal.

The parasites lie in the seams of the underclothing in which they lay their eggs. The eggs hatch in 7 days and reach the adult stage in about 3 weeks. At this stage they leave the clothing to seek food in the skin.

Clinically the disease is evidenced by marked itching and parallel scratch marks across the upper half of the back about the abdomen and the upper half of the buttocks. In chronic cases (vagabond's disease) there is a lichenification and pigmentation even furunculosis or pyoderma from scratching and rubbing.

Differential diagnosis must be made from urticaria by the absence of wheals and the typical localization of the scratch marks also from senile pruritus by examination of the underwear for pediculi.

Treatment. (1) All clothing must be removed and sterilized by steam or heat. The bedclothes must be laundered and ironed. (2) The patient takes a 1:10,000 bichloride of mercury bath in the morning to kill those parasites attached to the lanugo hairs.

(3) At bedtime one of the antiscabietic ointments is applied over the entire body from the neck down once daily for 3 days. (4) For any residual itching a phenolated calamine and zinc liniment is prescribed.

DDT (dichloro-diphenyl-trichloro-ethane) an insect repellent drug is applied as a spray or a dust to the clothing which should

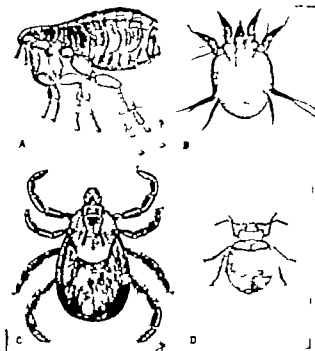


FIG. 74 (A) Ct. flea (B) Scabies mite (C) Wood tick (D) Bedbug (Burra f Entomology U S Dept of Agriculture)

be worn for 10 days before discarding. It is valuable in the mass treatment of pediculosis and other animal parasitic infestations.

Prophylaxis. Pyrethrum powder or flowers of sulfur should be sprinkled in the clothing and the bedclothes. frequent bathing and change of bed sheets and clothing are important. suspicious sleeping quarters and bed partners should be avoided.

PEDICULOSIS PUBIS

Pediculosis pubis is marked by the presence of crab lice in the hairs of the pubic, the axillary the abdominal and the perianal regions the chest and the limbs in hairy patients and in the eyebrows and the eyelashes, which may cause blepharitis especially in children.

The clinical symptoms are itching and a biting sensation occurring at irregular intervals. In old cases bluish-gray stains, *maculae ceruleae* and tiny excoriations are present.

Etiology Most cases of pediculosis pubis are acquired through sexual intercourse others may be contracted from dirty towels. Children may contract the disease from the maid or from some other member of the household. The disease is a vector for granuloma inguinale. The parasites are larger than those found in pediculosis capitis. They are found with the head buried in the follicle the claws clutching the hair and are visible to the naked eye.

Diagnosis is made by the presence of pubic itching without any evidence of dermatitis. In the examination the parasites should be looked for at the base of the hairs.

Differential Diagnosis. Public itching also occurs in diabetes and tinea cruris. Reflex itching sometimes accompanies bladder or prostate conditions.

Treatment. All hairy parts of the body except the head should be treated thoroughly. The hair should not be shaved nor should blue ointment or crude oil be used because of the danger or dermatitis.

The following preparations are employed (1) Phenolated camphor is preferable and should be applied locally for 3 days. After the third day dilute acetic acid or vinegar is applied to remove the nits from the hairs (2) Ammoniated mercury ointment is useful but may produce folliculitis. (3) Cuprex (Merck) is a complex parasiticide containing tetralin acetone and copper oleate. It is applied locally reapplied after 10 minutes and then washed off with soap and water after one hour. Although a popular preparation, the solution frequently produces irritation on sensitive skins (4) Tinctures of larkspur or stavemore are clean but inefficient. (5) Bornex (Wyeth) is nongreasy and noninflammable and is often successful with one application (6) Ten per cent of DDT in talc or pyrophyllite also can be used effectively

(3) At bedtime one of the antiscabietic ointments is applied over the entire body from the neck down once daily for 3 days. (4) For any residual itching a phenolated calamine and zinc liniment is prescribed.

DDT (dichloro-diphenyl-trichloro-ethane) an insect-repellent drug is applied as a spray or a dust to the clothing which should

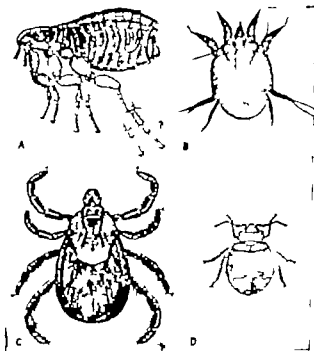


FIG. 75. (A) Cat flea. (B) Scabies mite. (C) Wood tick. (D) Bedbug. (Bureau of Entomology U. S. Dept. of Agriculture.)

be worn for 10 days before discarding. It is valuable in the mass treatment of pediculosis and other animal parasitic infestations.

Prophylaxis. Pyrethrum powder or flowers of sulfur should be sprinkled in the clothing and the bedclothes. Frequent bathing and change of bed sheets and clothing are important. Suspect sleeping quarters and bed partners should be avoided.

BEDBUG BITES

Bedbugs (*Cimex lectularis*) are world-wide in distribution. They are flat, rusty-brown parasites, which have an offensive odor and live in the crevices of beds, furniture walls and luggage. The eggs hatch in 7 to 10 days. The bugs are said to live 12 months without food. The ankles and the buttocks usually are attacked. Pruritic wheals with a central hemorrhagic spot soon appear although individual reactions may vary. Prurigo-nodularislike lesions may occur in the tropics. The bites usually appear in groupings of 2 sometimes 3 and persist for a week or longer. Infested rooms, beds, and crevices should be sprayed with 5 per cent DDT in kerosene.

Treatment. The bites should be sponged with 1 per cent camphor in alcohol or calamine and zinc-oxide lotion.

TICKS

Ticks are found on pine trees and in the underbrush. They may be acquired also from contact with infested dogs or cattle. Many patients do not realize they harbor a tick and suspect a wart. To remove the parasite touch it with a lighted cigarette or hot match tip and it will release its hold. In most cases, no local reaction occurs after the tick is removed. When ticks remain on the skin for several weeks, a chronic granuloma may develop which often resembles a lymphoma histologically.

CHIGGERS (RED BUGS)

Chiggers (*Trombicula irritans*) are a variety of tick which is distributed throughout the Southern and the Middle Western States. They infest the tall grass, the vegetation and the ground during the summer months.

The chigger first attacks the ankles and unless impeded by a garter will travel up the legs to the thighs and the trunk where the belt or the waistline puts an end to his wanderings. The mites firmly attach themselves to the skin and after engorging with blood fall off. There is an immediate burning sensation which is replaced by an intense pruritus. This continues for two weeks or more. Some individuals are immune to chigger bites.

Examination reveals several large excoriated edematous urticarial lesions.

Prophylaxis. Often chigger bites can be avoided by wearing proper clothing including shoes, stockings, puttees or long trousers.

ers. Powdered sulfur dusted on the feet and the legs is a useful form of prophylaxis.

Treatment. The indications for treatment are to relieve the pruritus prevent scratching and secondary infection. A good routine plan is to sponge the lesions with 70 per cent rubbing alcohol followed by an application of the following lotion

Phenol (Biquified)	
Campbor 2℥	0.5
Neocalamine and zinc oxide lotion	qs. ad 100.0

An oatmeal-baking soda bath at bedtime is soothing and promotes healing. Phenolated camphor colorless nail polish or colodion are useful in some cases.

Public health measures consist of removing the underbrush of the infested area, cutting the grass short and spraying the vegetation with sulfur or Chlordane (Velsicol Corp.) DDT is not effective.

DEMODEX FOLLICULORUM

Demodex folliculorum is an acarualike parasite found in the sebaceous follicles of the face. It is believed to have no pathologic significance in man although it frequently affects dogs. It may play a minor role in some cases of rosacea.

Treatment consists of the frequent use of soap and water followed by a 3 per cent sulfur ointment.

MOSQUITOES, BLISTER BEETLES AND GNATS

The local reaction is variable and may be urticarial, purpuric, bullous or eczematoid. Impetigo and abscesses may result from scratching.*

Prophylaxis consists of the use of fine copper screens netting over the beds and the spraying of rooms, but not the skin with Lindane, Toxaphene, DDT solutions or dust. Citronella oil and other mosquito repellents may produce a contact dermatitis if applied accidentally or directly to the skin. Dimethyl phthalate dabbed on the skin or applied as a 40 per cent cream is effective for several hours as a repellent.

* For a general discussion of the insect bite problem see Allington, H. V. and Allington, R. R. *Insect bites*, J.A.M.A. 155:240, 1954.

Treatment consists of the application of calamine and zinc lotion containing phenol or menthol.

MISCELLANEOUS PARASITES

Those to be considered include ants, caterpillars, spiders, beetles, scorpions, bees and wasps. The eruption consists of a localized group of bullae surrounded by an area of brawny edema which is treated on general principles.

Mites may also cause industrial dermatoses. Poultry handlers, harvesters, longshoremen, farmers and berry pickers are sometimes infested with mites from contact with mite-infested products.*



FIG. 74 Larva migrans (sandworm disease)

CREEPING ERUPTION

Creeping eruption (larva migrans) consists of progressive tortuous, threadlike lesions produced by the burrowings of filariform larvae of flies or nematode worms. The infestation has a world wide distribution and may be caused by (1) *Gastrophilus* (2) *Hypoderma bovis* or (3) *Ancylostoma braziliense*. The larvae are

*See Booth, B. H. and Jones, R. W. Mites in Industry A.M.A. Arch. Dermat. & Syph. 59:531 1954

found in warm sand contaminated by the excreta of dogs and cats. The disease is common in Florida and Texas and along the lower eastern coastal regions. The characteristic eruption consists of tortuous red linear lesions interspaced with tiny papules. Bathers acquire the disease by lying on the wet sand. The parasite is found just ahead of the active end of the lesion.

Prophylaxis. Workmen should be fully clothed when working in damp sandy areas. Bathers should avoid unclean and unregulated beaches.

Preventive measures include (1) exclusion of dogs and cats from public beaches, (2) spraying polluted sand piles with larvicide, (3) supplying fresh sand for children's sand piles, and (4) cauterization of suspicious insect bites.

Treatment consists of freezing the areas with an ethyl-chloride spray or solid carbon dioxide (30 seconds). Bermuda-onion poultices are often curative. Stibranose may be useful in the generalized cases. In resistant cases Hetrazan (diethylcarbamazine) may be used in doses from 0.5 to 2.0 mg per Kg body weight 3 times daily for from 1 to 3 weeks.

SCHISTOSOME DERMATITIS (SWIMMER'S ITCH)

This is caused by larval flukes which live on fresh water snails. The infestation is limited to the skin. The condition is fairly common in swimmers and fishermen in the sluggish lake region of upper Wisconsin and Michigan. It is characterized by a pruritic papular eruption on the feet and the legs. Vigorous wiping immediately after coming from infested water is a useful prophylactic measure. Cases should be reported to the state and the federal health authorities.

Treatment consists of applications of phenolated neocalamie lotion to the itching areas.

ENDOPARASITES

RAT BITE FEVER

This blood infection is produced by the bites of rats and other rodents and is caused by the *Spirochaeta morsus moris*. The Japanese type is known as sodoku. The disease is characterized by paroxysms of intermittent fever, generalized adenopathy and an eruption of erythematous macules on the abdomen, the neck

and the chest, resembling the rose spots of typhoid. Later these macules become erythematous, indurated plaques. There may be an intense inflammation at the site of the bite with accompanying secondary erysipelas or lymphangitis.

Course. Spontaneous cure is the rule in the well-nourished. Arthritis and endocarditis are complications. Death may result from endocarditis or septicemia.

Treatment consists of cauterization of the bites with phenol or nitric acid as a prophylactic measure. Penicillin is the drug of choice. The dose is 600,000 units of penicillin daily for 4 days. Terramycin is also effective.

NURSING ASPECTS

Pediculosis and scabies are the only members of the group which are contagious. Contaminated sheets used in the examination should be discarded at once, and the nurse will lose no time in washing her hands, for psychological reasons at the least.

Pediculosis Capitis. These cases usually are discovered by the school nurse. Severe itching, eczematous or impetiginous areas and enlarged cervical and occipital glands are always suspicious signs. Using tongue depressors to separate the hairs, the active parasites and the ova (nits) can be observed over the ears or in the occipital area. In doubtful cases, a pediculus may be removed from the hair and placed on a clean glass slide for microscopic examination. Various proprietary ointments are available as well as the old crude-oil treatment. In private practice, the diagnosis always should be withheld from the patient unless the parasite can be demonstrated to his satisfaction. It never is advisable to acquaint neurotic or fastidious women with the diagnosis otherwise the physician loses a patient.

Pediculosis Corporis. These cases occur frequently in patients in municipal clinics. Since the parasites live in the clothing which is removed and sterilized in the receiving room there is no danger to the nurse who cares for the patient. Admission is usually for the treatment of secondary infections (boils, impetigo, cellulitis, etc.)

Scabies. If the history indicates scabies, the physician will instruct the nurse to have the patient strip but not to have him lie on the table so that danger of contagion is minimized. If localized pyodermae are present the nurse should wash her hands thoroughly after dressing the lesions.

Infected school children may be readmitted to school after a thorough 24-hour application of one of the standard antiscabietics. Social workers should check other members of the family for the presence of the disease.

Diseases Due to Filtrable Viruses

THE HERPES GROUP

HERPES SIMPLEX

KAPOSI'S VARICELLIFORM

ERUPTION

GENITAL HERPES

HERPES ZOSTER

VERRUCA

VERRUCA VULGARIS

"VENEREAL WARTS

PLANTAR WARTS

FLAT JUVENILE WARTS

DIGITATE WARTS

FILIFORM WARTS

MOLLUSCUM CONTAGIOSUM

LYMPHOGRANULOMA

VENEREUM

NURSING ASPECTS

AN INCREASED number of heretofore unsolved skin diseases have been found to be caused by a virus. In some cases, the direct etiology is questionable in others filtrable fine granular bodies (granulosa) have been discovered in the lesions themselves. Those which affect the epidermis are dermatropic those which attack the central nervous system are neurotropic.

Advances in virus research have been made by (1) cultivation of viruses on inoculated chick egg (chorio-allantoic membrane) (2) darkfield studies, (3) demonstration through fluorescence, (4) inoculation into rabbit's cornea, (5) biopsy and special stains, (6) antibody studies and (7) electronic microscopic studies*.

THE HERPES GROUP

HERPES SIMPLEX

Herpes simplex is an acute eruption characterized by grouped small superficial vesicles on the lips, the cheeks, the face the fingers the buttocks. It is caused by a specific virus activated by various trigger excitants.

Clinical Description. There is usually a history of sudden onset. In some cases a gastric disturbance or a cold precedes the

For an excellent review of the subject see Hilsenan, M. R. Viruses of special interest to the dermatologist. *A.B.A. Arch. Dermat. & Syph.* 61:210, 1950, and Blank, Harvey and Rahn, Geoffrey. *Viral and Rickettsial Diseases of the Skin, E. and Mucous Membranes of Man*, Boston, Little Brown, 1955.

eruption. In a typical case one observes a group of superficial small vesicles on a reddened base. When the vesicles occur on



FIG. 75 Herpes simplex. The vesicles are closely grouped on an erythematous base; they contain lemon-colored serum and persist for from 6 to 10 days during which antibodies develop.

the mucous membranes of the lip or the buccal mucosa they tend to rupture easily and to leave superficial ulcers. After a day or so the ruptured vesicles become covered with a typical thin straw-colored crust. The lymph nodes may be enlarged and tender. No scarring results after the lesions have completely involuted unless complicated by secondary infection.

Recurrences may appear on the same site several times annually as a result of reactivation of the virus in the tissues by nonspecific stimuli.

Etiology. The disease is common in all ages but is most likely to affect children. When the disease occurs in the mouth it is known as herpetic stomatitis.

THE PREDISPOSING FACTORS are those which lower the resistance of the affected part, permitting the virus to injure the

sensory nerve endings. The disease may follow emotional disturbances, local anesthesia, artificial fever therapy or fevers including colds, natural or induced malaria, pneumonia, cerebrospinal fever etc. In predisposed individuals herpes simplex occurs on the face or the lips following exposure to the sun or on the buttock or the inner thigh preceding or during menstruation. Recurrent cases may be due to reflex irritation from an impacted tooth or cerumen in the ear canal.

THE EXCITING CAUSE is a neurotropic virus which is distinct from that which produces genital herpes or herpes zoster. Apparently the virus is more virulent than those which produce other types of herpes. Inoculation produces keratitis and meningo-encephalitis in experimental animals. About 50 per cent of all individuals are carriers of this virus.

Differential Diagnosis. Herpes simplex sometimes is confused with impetigo contagiosa. In the latter disease new lesions form more rapidly the individual lesions are more extensive the bullae are flaccid and not tense as in herpes and the crusts are honey-colored and not straw-colored. Herpes zoster when it occurs on the face may be mistaken for herpes simplex. The lesions of zoster often follow the course of a branch of the fifth cranial nerve usually the supra-orbital nerve. The vesicles are more grouped often hemorrhagic, preceded by pain and characterized by a unilateral distribution.

Diagnosis in doubtful cases is made by Paul's test (inoculation on a rabbit's cornea) biopsy with special staining (inclusion bodies) cytologic wet smear studies (large number of mononucleated and multinucleated balloon cells) and agglutination tests.

✓Treatment. In the ordinary case, the routine treatment consists of the local application of mild astringents. The following prescription is useful

Camphor	3.0
Menthol	0.25
Alcohol (60%)	100.0

Phenolated camphor camphor spirit or a styptic pencil applied 3 or 4 times a day and zinc oxide ointment at night is a good routine procedure. Extensive cases often respond to intramuscular injections of from 5 to 10 cc. of blood 2 or 3 times a week. In resistant cases roentgen irradiation (50 r at weekly intervals for 3 weeks by an experienced dermatologist) may be of benefit al-

though if effective the result may be psychological. Puncture vaccination with smallpox virus has its advocates, but there is no scientific basis for its use because there is no cross immunity between the herpetic and the vaccinia viruses. Cortisone and ACTH should be withheld as they tend to disseminate the infection.

Recurrent Cases. One or more of the following methods may reduce the frequency of attacks or cure the condition: (1) removal of the provocative cause (2) attention to psychosomatic trigger influences (3) modification of the soil by low uric acid or carbohydrate diet (4) therapeutic shock (autobemotherapy) (5) reduction of virulence of the virus (x ray therapy to lesion) (6) chemotherapy (hexamethylene or trypan blue) or (7) vaccination (intradermal injections of serum from vesicles at 5-day intervals).

KAPOSI'S VARICELLIFORM ERUPTION (ECZEMA HERPETICUM)

This rare type of generalized herpetic infection may occur in patients with an exudative diathesis, especially atopic eczema.

Clinical Symptoms. After an incubation period of 8 days the onset is acute with fever, cervical and submaxillary adenopathy, rapidly spreading vesicles on the face and the nose which soon become umbilicated vesicopustules. The skin becomes acutely inflamed with edema and crusting. The eyes may become involved (keratoconjunctivitis or lid lesions).

Diagnosis. Serum from early unruptured vesicles should be inoculated in the cornea of a rabbit (Paul's test). Biopsy may reveal herpes inclusion bodies. A history of exposure to the herpes virus (fever blisters) may be obtained.

Etiology. The cause is the virus of herpes simplex and in some cases of the virus of vaccinia. Infants and young adults usually are affected.

Differential Diagnosis. Vaccinia, poison-ivy dermatitis, impetigo and must be excluded.

Prognosis. The disease may be fatal in infants and children.

Treatment consist of antiseptic wet packs, small pox penicillin or Chlormycetin therapy, intravenous fluids, blood transfusions and adequate nursing care.



FIG. 76 Kaposi's varicelliform eruption (Blattner R. J., Heyls, F. M. and Harrison, M. K. The etiology of Kaposi's varicelliform eruption. *J. Pediat.* 27: 207)

GENITAL HERPES

Genital herpes is a variety of herpes simplex consisting of very small grouped vesicles with thin roofs on a reddened base in a patient with an anxiety complex. When the vesicle ruptures it leaves a superficial ulcer with a polycyclic border

The subjective symptoms consist of itching and burning. The eruption may appear on the glans, the sulcus or the prepuce in women on the vulva or the vagina. This type of herpes often is mistaken for a venereal disease. The lesion may act as a portal of entry for the *Treponema pallidum* or the Ducrey bacillus.

In many of the cases the characteristic features of the disease are disguised by an inflammatory reaction resulting from self treatment with tincture of iodine caustics Mercurochrome or various powders.



FIG. 7. Genital herpes. The vesicles are tiny and tense. They rupture to form superficial ulcers with polycyclic borders. The prepuce and the balanopreputial groove are more common sites.

Etiology The predisposing factors which lower the resistance of the skin to infection include catheterization, masturbation, pregnancy, menses, cystitis, focal infection and painful colitis. It seems probable that patients may be carriers without having the actual disease. Since there is no lasting immunity, recurrent attacks are common.

Differential Diagnosis. Herpes genitalis may be confused with chancroid, but the history of previous attacks, shorter incubation period, absence of inflammatory reaction and ulceration and freedom from inguinal adenitis distinguish herpes from this condition. Chancres are more indurated, the incubation period is longer and the darkfield is positive even though the serology may be negative.

Pathology Histologic examination shows acidophilic intranuclear bodies within the epithelial cells. There is also marked intercellular and intracellular edema.

Prognosis. If the trigger factors can be discovered and removed, spontaneous recovery occurs. Otherwise the lesions may recur over a long period of time in spite of therapeutic measures.

Treatment. Although the layman is addicted to the use of strong topical remedies, the physician should employ soothing remedies, including thymol iodide powder or 1 per cent silver nitrate. In every case the blood should be examined for evidence of syphilis. If any suspicion exists, regardless of the history, a darkfield examination also should be made at the time of the first visit. Repeated smallpox vaccinations may help. Circumcision is advised if everything else fails.

HERPES ZOSTER

Herpes zoster is an acute trophic manifestation of a ganglionitis characterized by grouped vesicles arranged along the course of a sensory nerve and associated with inflammatory changes in the corresponding posterior spinal ganglion.

Clinical Description. The disease, which is a zonal dermatosis, consists of grouped papules, papulovesicles or vesicles on an erythematous base, arranged along the cutaneous distribution of a sensory nerve with the lumbar or the thoracic segments affected in 75 per cent of the cases. When the disease is severe, ulceration and even gangrene may result. The disease is practically always unilateral. Only one spinal segment usually is involved. Rarely two contiguous segments may be affected.

In aged and enfeebled patients with zonal zoster aberrant lesions may occur which often are confused with varicella. These cases are known as herpes zoster generalisatus. There is no agreement among authorities concerning the nature of this rare type. Some regard the virus of both diseases as being identical others feel that the infection may overflow in the posterior horns of the cord and affect other segments.

Motor nerve involvement occurs in about 1 per cent of the cases and usually is associated with the trigeminal nerve types (facial paralysis). Transient paralyses of the arms or the legs may occur in children.



FIG. 78 Typical location of herpes zoster (From Dr. V. Pardo-Cast. Do)

BILATERAL HERPES ZOSTER is rare. It differs from the usual infectious type in that there is an obvious cause present, such as trauma, lymphoblastoma or metastatic carcinoma.

Stages of the Disease. IN THE PRE ERUPTIVE STAGE the onset is gradual with mild moderate or very severe pain or "burning sensation along the involved nerve. In this stage the disease may be mistaken for neuralgia, angina pectoris, gallbladder disease, renal colic and other acute surgical conditions.

THE ERUPTIVE STAGE, which appears about 3 days later, is ushered in with an eruption of typical grouped vesicles. These are tense, pinhead size to lima-bean size, oval round or irregular containing clear or hemorrhagic serum.



FIG. 79 Herpes zoster with involvement of the first branch of the trigeminal nerve. The nasal branch was not affected, so that the cornea escaped.

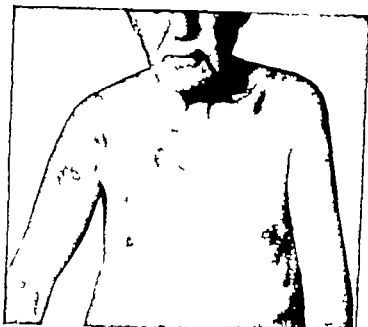


FIG. 80 Herpes zoster in a typical location. The eruption was preceded by neuralgic pains and was followed by residual pigmentation.

In the abortive cases the eruption consists of grouped red maculopapules. In the severe cases especially the facial types, the vesicles may become hemorrhagic, or actual ulceration may result.

THE POSTERUPTIVE STAGE (postherpetic neuritis) may be short or may last for a year or longer. It usually is more severe in the aged and the debilitated. Following the disappearance of the eruption the patient complains of moderate mild or severe pain which is due to a neuritis of the involved nerve. The pain is severe enough in some cases to necessitate the use of narcotics.

Etiology THE PREDISPOSING CAUSES are usually trauma deep x ray therapy exposure to cold or focal infection. Most cases occur in those over 40 years of age especially during the colder months.

THE EXCITING CAUSE is a neurotrophic virus that invades the ganglia of the roots of the spinal nerves and occasionally the cranial nerves.

Pathology The outstanding lesions are multifocal vesicles with their bases lined with "balloon cells" or vacuoles. The

epithelial cells usually contain inclusion bodies. There is a leukocytic infiltration in the corium and intercellular and intracellular edema of the epithelial cells.

Differential Diagnosis. The localized pain in the pre-eruptive stage may be mistaken for neuritis and the erythema preceding the vesicular eruption for a contact dermatitis, but these errors usually are made by the patient. Where a heating pad has been used for the neuritis, a burn may be suspected but after a few days, the true diagnosis is evident.

Prognosis. The average case on the trunk or an extremity usually clears up within three weeks. When the supra-orbital or the ophthalmic nerves are affected serious eye complications may occur.

Treatment. Although the disease is self limited the purpose of treatment is to prevent extension of the disease, to reduce the incidence of complications and relieve the patient of the concomitant symptoms. No specific antiviral agent is known at this time. The physician should be wary of employing new drugs in this disease unless scientific controls have been made to evaluate their true effect.

All extensive cases and those cases occurring in the senile should be hospitalized if possible. Consultation with an oculist should be obtained in all facial cases with eye involvement.

✓**LOCAL TREATMENT** is important in order to protect the parts from irritation friction and infection. This may be accomplished by the use of thick cotton pads saturated with zinc stearate powder the application of flexible collodion or calamine type lotions.

CONTROL OF PAIN In the average case, aspirin or phenacetin is effective. Opiates may be necessary in extensive cases and in the senile.

INJECTION THERAPY While local treatment is usually sufficient for the ordinary case collateral treatment is often necessary although it is difficult to prove that parenteral therapy will shorten the course of the disease. The injection of posterior pituitary extract in doses of 1 cc. on alternate days relieves the pain and shortens the course of the disease. Sodium iodide is my treatment of choice because of its effectiveness and dependability. The drug (gr 31/2) is given intravenously daily for 7 days.

In cases that do not respond to sodium iodide autohemotherapy is sometimes successful. From 5 to 10 cc. of blood is injected into



FIG. 81. Verrucae vulgares in an unusual location. (From Dr. Beatrice H. Kuhn)

the buttock twice weekly. We have not seen any beneficial results from Protamids—a colloidal solution of a proteolytic enzyme. The same can be said for B₁₂, although it may improve the patient's resistance. Intravenous injections of procaine and thiamine and intramuscular injections of Etamon may be useful in some cases.

ANTIBIOTIC THERAPY. Aureomycin appears to lessen the duration of an attack when given for 3 or more days, although a negligible amount of the drug enters the cerebrospinal fluid.

POSTHERPETIC NEURITIS is treated with radiant heat diathermy, x-ray irradiation over the affected ganglion and anodyne drugs. In cases associated with persistent intractable pain infiltration of the corresponding ganglion with procaine hydrochloride, section of the posterior nerve roots, cordotomy or total excision of the involved dermatome may be useful.

VERRUCAE

Etiology. Warts are unstable epithelial growths characterized by prickle-cell layer proliferation (acanthosis) and caused by a filtrable virus. They are auto-inoculable in nature. The various types of warts are probably due to the same virus but a difference in virulence and anatomic location produces a varied clinical picture. Although local and general immunity often occur follow

log the disappearance of warts this is by no means true in all cases.

The term "papilloma" is too indefinite clinically and should not be used to designate a wart.

A wart is produced by a stimulus from the virus which stimulates mitosis in the prickle-cell layer. This causes extensive proliferation with branching, thus giving rise to warty projections.

The diagnosis of a wart is relatively simple. In difficult cases a hand lens should be used to demonstrate the warty surface. Since warts occasionally may resemble squamous-cell carcinoma or keratoses, a biopsy should be made in such cases to verify the diagnosis.

Treatment. There is no specific therapy. Some warts respond to relatively simple measures, while others resist the most energetic therapy. It is difficult to evaluate those measures which are successful in a given case because of the effect of secondary influences. Even the action of x ray therapy on warts is considered by some to be purely psychological.

VERRUCA VULGARIS

The common type of verruca is usually grayish in color, pin-head size to pea size and has a warty surface. There is no surrounding inflammation present unless complicated by infection. In some cases painful fissures may occur especially in winter. Warts may persist for several years without any change in their appearance.

The lesions are often multiple from reinfection. While warts usually appear on the dorsum of the fingers and the hands, they occasionally are observed in the scalp, the nostrils, the lip, the subungual spaces or the nail folds.

Etiology. There apparently is a wide variation in individual susceptibility. The incubation period may vary from 4 weeks to 6 months. Occupational causes include chronic exposure to tar pitch and crude petroleum. Multiple warts especially those on the fingers and hands are commonly seen in children and adolescents. Warts on the knees often follow mild trauma which probably favors implantation of the virus.

Pathology. There is a distinctive picture: a marked hyperkeratosis, granulosis, acanthosis with deep rete pegs often bent toward the center of the growth and mitoses in the cells of the prickle layer.

Differential Diagnosis Squamous-cell carcinoma and beryllium sarcoid must be considered.

Prognosis Warts may persist for several years. They may disappear spontaneously or after trauma or local infection. Some patients "bite them off" a method not without danger.

Treatment. Warts vary in their response to treatment. It is best to use conservative measures first. In cases of multiple warts treatment of one lesion may cause the others to disappear.

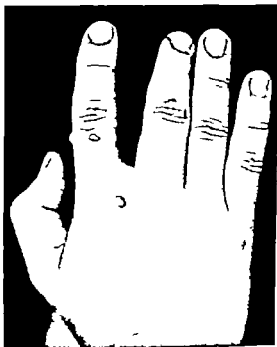


FIG. 82. *Verrucae vulgaris* in a common location. These early lesions were destroyed by fulguration and curet.

Acids. The simplest treatment is the use of acids. However there is danger of disfiguring scars or even keloids. When using ✓glacial acetic, dichloroacetic or trichloroacetic acid the normal skin should be protected with a ring of petrolatum. If the wart is a large one it can be curetted first and the base carefully treated with the acid.

Solid carbon dioxide, liquid oxygen or nitrogen also may be used, especially in the subungual type the application requiring from 15 to 60 seconds.

CAUTERY If the wart is on a covered area, it may be destroyed by surgical diathermy or the electric cautery. The lesion is anesthetized with 2 per cent procaine solution and the wart destroyed to its base. Warts destroyed by surgical diathermy require a much longer time to heal.

X RAY AND RADIUM when prescribed by the experienced dermatologist are the preferred methods if the lesion is under 1 year old, because (1) treatment is painless (2) no dressings are required, and (3) there is little or no scarring. This type of treatment is preferred in treating subungual lesions. Radium therapy also is preferred in children who may resist the more radical methods.

SURGICAL EXCISION is not advisable because of the frequency of recurrences and the size of the resultant scar.

OTHER TREATMENTS Oral bismuth is useful in the generalized cases in children but its effect is erratic. The dose is one tablet after meals, gradually increased to 3 if tolerated. Treatment is continued for at least 3 months, or less if results are obtained.

The value of psychotherapy in the treatment of warts is still controversial. This type of therapy is based on the theory that nerve impulses introduced through the emotions in susceptible subjects may cause physicochemical or biologic changes in the tissues, thus damaging the virus.

Vaccine therapy may be useful when all other methods have failed. The wart is macerated in normal saline, run through a Berkefeld filter and the solution sterilized. The dose of the filtrate is 0.1 cc. injected intradermally twice a week.

"VENEREAL" WARTS

These sometimes are called fig warts and are infectious. The term "*verruca acuminata*" is preferable since the descriptive adjective "venereal" is unfair in many cases because of its implications.

Clinical Description. These are gray bluish or pink, filiform warts bunched together and forming cauliflowerlike growths. The individual lesions are the size of a split pea. In women extensive areas of the vulva may be involved with the production of large vegetating masses. A foul-smelling exudate is sometimes

Diagnosis. The condition may be mistaken for lichen planus, but the absence of a violaceous color, mouth involvement and itching aids in the diagnosis. In doubtful cases a biopsy is helpful. In papular acne the lesions are not lichenoid, do not appear in scratch marks and are associated with comedones and more or less inflammation.

Treatment. Juvenile warts are usually resistant to treatment. In some cases several months are required. The local treatment consists of the use of mild exfoliating applications (30 per cent sulfur ointment, half strength Vlemmink's solution and lotio alba. If irritation develops, soothing ointments should be employed to allay the inflammation.

Mercury protoiodide may be prescribed in doses of from gr $\frac{3}{8}$ to gr $\frac{1}{4}$ twice a day for at least 2 months. If diarrhea occurs, the dose should be cut down.

Aureomycin cream may be effective in some cases. Destructive methods are not advised because (1) there is danger of scarring, and (2) the warts often disappear of their own accord.

Psychotherapy (suggestion) is more apt to be effective in this type of wart. Thorium X varnish applied every 4 weeks for several weeks may be useful also.

DIGITATE WARTS

Digitate warts are clusters of fingerlike warty projections on an oval or round base. They bleed easily following trauma. They are usually multiple and are found on the scalp, the beard, the nostril, the tongue or the neck.

Treatment. Fulguration is the ideal form of therapy.

FILIFORM WARTS

These are small threadlike flesh-colored or light-brownish lesions, with a cornified tip usually occurring on the neck, the eyelids, the anterior axillary folds or the nostrils.

Treatment, which is relatively simple, consists of snipping off the lesion with sterile scissors and destroying the base with the cautery.

MOLLUSCUM CONTAGIOSUM

This disease consists of an eruption of small discrete pearly or waxy globular white or pink tumors with umbilicated centers, a central dark plug and a tendency to occur in scratch marks. If

Infection is present as a result of irritation or scratching the lesion is surrounded by an inflammatory area and is tender to the touch. These growths, which may appear anywhere, except the palms and the soles, usually are found on the trunk and the buttocks they are rare on the face but may occur on the eyelids and the genitalia *

Etiology The disease commonly is acquired on playgrounds, in schools, swimming pools etc. The incubation period is from 6 to 12 weeks. The condition usually occurs in children and rarely in adults. The exciting cause is a dermatropic virus.

Pathology consists of a hypertrophy of the epidermis and rete with dyskeratosis (vacuolization and inclusion bodies) and hyalinization of the central cells, thus forming the molluscum body. The capsule of the lesion is formed by compressed connective-tissue cells.

Prognosis. If untreated the lesions multiply by reinoculation and persist indefinitely.

Treatment. The standard treatment consists of making a superficial incision through the roof of each lesion with a sharp-pointed knife and expressing the cheesy contents. Bleeding may be stopped by compression. This type of treatment is not followed by scarring. In unmanageable children, a short general anesthetic in a dental office will permit the operator to work quickly and skillfully. Infected lesions first should be treated with antiseptic wet dressings.

Aureomycin or Terramycin by mouth may be used in the generalized cases but results are erratic and unpredictable.

It is important to examine the entire body carefully in order not to miss any lesions. Following treatment, the clothing should be sterilized to prevent recurrence. Other members of the family also should be examined.

LYMPHOGRANULOMA VENEREUM

This, the genitoretal syndrome is a venereal disease caused by a filtrable lymphotropic virus and is characterized by three stages (1) primary or local infection (2) involvement of the superficial and the deep inguinal lymph vessels and nodes and (3) late cicatricial changes in the rectum and the surrounding tissues.

For an excellent review of the subject see Low R. C. Molluscum contagiosum, Edinburgh M J 53.657 1946

Clinical types (1) anorectal (2) inguinal, (3) pelvic, (4) genital (5) mixed and (6) asymptomatic.

Clinical Description. The incubation period varies from 3 days to 3 weeks. The disease begins as a benign evanescent papule, vesicle or pustule which may be situated on any part of the genitalia. Intra urethral and extragenital lesions are not uncommon.

The inguinal adenitis appears after a period of 4 weeks with involvement usually limited to one side. The nodes are matted together and are adherent to the overlying violaceous skin. After a few weeks they break down with the formation of fistulous sinuses.

IN MALES elephantiasis of the penis or the scrotum may follow extensive involvement.

IN THE FEMALE the course is different because of the perirectal lymph drainage in this sex. Inguinal adenitis is rare in



FIG. 86 *Lymphogranuloma venereum* (inguinal type) \ primary lesion was observed (Dr L M Smith's case)

women but symptoms referable to the rectum are common. Eventually inflammatory proctitis occurs ending in tubular or annular strictures. Elephantiasis of the vulva may follow as a sequela in some cases.

Moderate secondary anemia and leukocytosis, hyperproteinemia and a transitory false positive V D R. L. reaction are not infrequent in the late cases.

Systemic disturbances are not uncommon and consist of fever, malaise, arthritis and toxic erythematous eruptions (erythema nodosum, erythema multiforme).

Etiology. The disease is caused by a filtrable virus, *the lymphogranulomatosis* acquired through sexual intercourse. Negroes especially are predisposed. Most cases naturally occur in the age group of greatest sexual activity. The latent and the abortive types may also be infectious.

Pathology. The lesion is an infectious granuloma. The glands are honeycombed with fistulous tracts. The overlying skin is attached to the swollen lymph nodes by fibrous adhesions.

Differential Diagnosis

	<i>Chancroid</i>	<i>Lymphogranuloma</i>	<i>Granuloma Inguinale</i>
Bacteriology	<i>Streptobacillus of Dugrey</i>	Virus	Donovan's inclusion bodies
Systemic disturbances	None	Yes	Absent
Frel test	Negative	Positive	Negative
Ulceration	Superficial	Suppuration	Deep and extensive with vegetations and granulomas
Adenitis	Present in 30% of cases	Characteristic	None
Pain	Yes	No	No
Incubation period	About 3 days	10 days to 3 weeks	Long

Diagnosis. A specific diagnosis is made by means of the Frel test, a Lygranum or a complement-fixation reaction when titers of 1:40 and above are obtained. A positive Frel test, however, does not necessarily establish the diagnosis, nor does a negative test exclude it. The same may be said of the complement fixation test. They are of definite confirmatory value in all cases where syphilis has been excluded.

Treatment. Bed rest is important in all acute cases to prevent extension of the disease. In the early cases sulfonamides and Aureomycin or chlormycetin are equally effective. In extensive cases excision of the affected glands should be considered. When colostomy is indicated in the lower bowel types a course of antibiotics should precede the operation.

The anemia should be controlled with iron and liver by mouth or injection. Intravenous fluids are indicated if electrolyte imbalance is present. A high protein diet is advisable.*

NURSING ASPECTS

The virus group of dermatoses includes "fever blisters," "shingles," warts and the venereal disease lymphogranuloma venereum.

Herpes Simplex. The nurse's duties include taking the patient's temperature, gently removing crusts and setting out a syringe for autobemotherapy and smallpox vaccine. Patients in hospital wards with atopic eczema must not be exposed to this infection.

Herpes Zoster. Elderly patients in pain should be handled as gently as possible. Since the affected area is usually very sensitive to pressure dressings should be removed carefully. Lotions or powders rather than ointments are employed to dry up the blisters. A thick pad of absorbent cotton is applied over the area to protect it from friction from the clothing and to avoid chilling. The extent of the eruption should be noted as well as any weakness of muscular groups.

Warts. Common warts may be infectious unless immunity is present so that it is best not to touch the lesion with the fingers. After fulguration the wound is painted with gentian violet, and an antibiotic ointment is applied to prevent infection.

VENEREAL WARTS are painted with a solution of podophyllin and talc powder is dusted on the area.

FLAT JUVENILE WARTS are treated by using a mixture of psychotherapy and bismuth pills.

Lymphogranuloma Venereum. The patient should be stripped and the routine examination to rule out syphilis, should be carried out. Frei antigen and rectal specula should be available.

*For details of therapy Greenblatt, R. B. et al. The newer antibiotics in the therapy of the venereal diseases other than syphilis. *J. Ven. Dis. Inform.* 31:45-50, 1950.

Diseases Due to Psychosomatic and Nerve Disorders

DERMATOPHOBIAS

ACAROPHOBIA

CANCERPHOBIA

NEUROSIS

PSYCHOGENIC DERMATOSES

DERMATITIS FACTITIA

ACUTE DISSEMINATED

NEURODERMATITIS

COMPULSION NEUROSES

NEUROTIC EXCORIATIONS

PRURITUS

GENERAL PRURITUS

LOCALIZED FORMS OF

PRURITUS

LOCALIZED NEURODERMATITIS

NURSING ASPECTS

Introduction. Since the skin is a powerful organ of emotional expression, the psychic element plays an important part in many disturbances of the skin although many patients are reluctant to concede a psychogenic factor. The emotions of fear, anxiety, personality defects, conflicts, repression and inferiority may be sublimated into stimuli affecting various parts of the skin. These stimuli may disturb the vasomotor mechanism through the autonomic nervous system with the production of urticaria or rosacea. If the sensory nerves are involved, pruritus may result. Nerve impulses also can be transmitted to the secretory glands resulting in hyperhidrosis or pompholyx.

The visual skin occupies an important place in the psyche of an individual. A healthy skin is an organ of expression denoting youth, vigor, vitality and cleanliness. Even minor deviations from the normal in some individuals (somatopsychic) may traumatize the psyche, (e.g. a few superfluous hairs). For this reason physicians should take as much interest in a minor dermatologic case as in one of major importance.

Although almost every skin disorder is associated with a normal amount of anxiety and apprehension, physicians should avoid blaming eruptions they are unable to diagnose on "nerves."

In the psychosomatic dermatoses one or more of the following neurotic symptoms may be present: headache, palpitation, hyperhidrosis, tightness in the chest, tremors, tenseness, urinary frequency, insomnia, numbness, fatigue and dysphagia.*

DERMATOPHOBIA

The phobias are superficial expressions of disturbed emotional states, the cutaneous reactions being relatively unimportant. The type of fear may have some close relation to the inner conflict although the patient does not realize it.

Most phobias are characterized by a feeling of distrust toward medical advice as well as a feeling of unworthiness.

ACAROPHOBIA (DELUSION OF PARASITOSIS)

Acarophobia, or fear of infestation with insects, is a paranoid condition ("mental itching") in which the patient insists that his skin is contaminated with parasites. He persists in his belief regardless of advice and frequently produces a piece of lint or epidermis, convinced that it is a parasite. Cerebral formication also occurs in cocaine addicts and in cerebral arteriosclerosis.

The prognosis is guarded. Treatment is a difficult problem since psychiatric consultation usually is refused. Aleahire (J.A.M.A. 155:15, 1954) cured 3 cases with an antipellagrous diet which was prescribed because of a history of nutritional deficiency. Electroshock therapy may be successful in some cases.

CANCERPHOBIA

Cancerphobia is the exaggerated fear of cancer which seems to be more prevalent in recent years following cancer campaigns. Undue anxiety may be felt toward Fordyce cysts of the lips, benign pigmented nevi, senile angiomas and geographic tongue.

LOCALIZED PSYCHOGENIC ERYTHEMA

Psychogenic causes acting reflexly through the vasomotor system may produce in susceptible individuals a recurrent erythema of the flush areas of the face as well as the chin, the upper chest and the upper third of the back.

Treatment consists of elimination of the psychogenic factor, antihistamines and sedatives (Barbitonina, Donnatal, Bellergal).

*For more detailed discussion of the relation of psychiatry to dermatology see Silberman, A. J. Psychiatric factors in dermatologic disorders. *Am. J. M. Sc.* 226:101, 1955.

NEUROSES

Since the skin is an organ of social importance neuroses may occur in relation to such minor skin ailments as superfluous hair enlarged pores, a few acne lesions and fancied alterations of the texture of the skin or the hair. These patients develop a fixed hypochondriasis and think they have lost status in the social world.

The dermatologic condition should be treated no matter how insignificant and an attempt made to build up the patient's other assets.

PSYCHOGENIC DERMATOSES*

DERMATITIS FACTITA

Dermatitis factita is an artificial dermatosis, or form of self mutilation produced by psychoneurotics, psychotics or frank malingerers through various chemical or mechanical means in an effort to obtain sympathy or for some other reason.

Clinical Signs. The eruption consists of sharply defined ulcerations, or areas of erythema or irregular bullae which usually assume a fantastic or artificial shape. The lesions are streaked if a fluid has been used the edges are jagged if a sharp instrument has been employed. The severity of the eruption depends on the nature and the strength of the agent used and the length of contact. Naturally the eruption occurs in areas accessible to the right hand in right handed individuals. The patient usually complains of exaggerated pain and burning but otherwise has a placid and innocent expression. Occasionally he may voluntarily predict the location of future lesions. The palate and the sclerae may be anesthetic, and the reflexes exaggerated.

Etiology This rare condition usually occurs in hysterical or psychotic young girls, many of whom are hyposensitive to self inflicted painful stimuli. In young patients, suppressed dramatic instinct maternal rejection or exhibitionism is often a predisposing cause. In older individuals, there is a desire for pity sympathy attention escape from unpleasant work, or compensation. The agents employed include caustic acids, alkalis and sharp instruments, in fact, anything that can be used to damage the skin.

*For an excellent discussion of the subject, consult Wittenber E., and Russell, B. Emotional Factors in Skin Diseases, New York, Hoeber 1953

Diagnosis is made by the bizarre nature of the lesions, the distribution, the unorthodox pattern and the unusual sequence of events. The history does not adequately explain the development of the lesion which usually clears up during periods of close observation and recurs during periods of stress. A study of the patient's attitude reveals smugness in the psychoneurotic, disinterest in the psychotic and hostility in the malingerer.

While the diagnosis may be easy for the expert, obtaining the proof may be extremely difficult.

Course and Prognosis. The disease appears suddenly and at irregular intervals, depending on the motivation. Outbreaks of the eruption continue until the cause is discovered and the patient admits defeat.

Treatment. The motivation must be analyzed and the situation adjusted. The patient's room should be searched for the causative agent. Then after the physician gains the patient's confidence, the facts should be presented with the object of obtaining a confession. Fixed dressings to prevent tampering with the lesions or the use of inked lines to measure off the eruption usually aid in a correct diagnosis and a rapid cure of the eruption. Sodium amytal, Evipal or hypnosis may be used to produce a state of partial narcosis during which the patient may admit guilt upon questioning.

ACUTE DISSEMINATED NEURODERMATITIS

This syndrome consists of a more or less acute dermatitis involving specific areas (eyelids, neck "V" of the chest and antecubital spaces) extreme itching and burning, and a state of nervousness, tension anxiety or despondency.

Etiology. The majority of cases occur in menopausal women who have been under a mental strain for some time (a death in the family an alcoholic or an unfaithful husband an unsatisfactory marriage of a son or a daughter etc.)

The condition may be considered as a psychosomatic allergy with choline dermal sensitization.

Treatment. Hospitalization is necessary for the severe cases where sedation and rest are important. Sedation with sodium amytal gr. 3 is advisable in disturbed patients phenobarbital, in the mild forms.

When the acute symptoms are under control one of the reserpine drugs (0.1 to 0.5 mg.) should be prescribed to reduce the

nervous tension. Steroid therapy for a week or longer if not contraindicated, may be very effective but results usually are temporary unless the basic cause is discovered and corrected. If the patient fails to get relief from steroid therapy intravenous procaine (0.1 to 0.2 per cent in isotonic normal saline solution) may give good immediate results. To avoid the possibility of procaine hypersensitivity a conjunctival or intradermal test must be made first.

Local therapy is not well tolerated but wet packs of Burow's solution in the moist cases and calamine liniment are often useful.

Prophylaxis. In order to prevent recurrences, the causal difficulty should be attempted by an adjustment of the patient's mental attitude, a conference with those involved in the situation or resignation to insolvable conditions with aid from religion or psychotherapy.

MISCELLANEOUS DERMATOSES

Dermatoses in which functional disturbances of the autonomic nervous system sometimes play an important part include alopecia areata, excoriated acne, rosacea, pompholyx, burning tongue, hyperhidrosis and atopic eczema. They are discussed under their appropriate headings. Psoriasis and seborrheic dermatitis may be aggravated by psychosomatic factors.

COMPULSION NEUROSES

NEUROTIC EXCORIATIONS

Neurotic excoriations are asymmetric traumatic eruptions in mentally disturbed patients due to an uncontrollable impulse to scratch or excoriate the skin. In spite of the fact that the skin may be covered with numerous traumatic excoriations they do not appear to be unduly disturbed by their presence.

Clinical Description. The eruption varies from scratch marks to ulcerations. Extensive excoriations may appear on the face, the neck, the chest or the limbs. There is an uncontrollable desire to pick, scratch, dig, pinch, rub or squeeze the skin. The palate and the sclerae are usually anesthetic, and the reflexes exaggerated.

Etiology. This is a compulsion neurosis with an underlying congenital environment, personality defect, tension make-up, anxiety neurosis or constitutional inadequacy. Mental disturbances may be sublimated into stimuli affecting the cutaneous

sensory nerves with resulting itching burning or stinging sensations. The patient obtains relief by a conversion of the intolerable itching sensation into a more tolerable pain sensation. In many cases the condition starts as a definite dermatosis of very *wild* character which may be transformed into neurotic excoriations by rubbing or scratching as a result of the development of an uncontrolled scratch-itch reflex. The patient compensates for his problem by literally "taking it out on his own skin."



FIG. 87 Neurotic excoriations are very common in institutional patients with chronic psychoses.

The large number of patients in our mental institutions who present this condition apparently derive a pleasurable sensation from scratching and are totally unaware of the damage they inflict on their skin.

Diagnosis. The excoriated appearance of the lesions is characteristic. The patient readily admits his guilt.

Differential Diagnosis. The condition must be differentiated from senile pruritus, scabies, pediculosis corporis, lichen planus and the lymphoblastomas.

Prognosis is poor unless the underlying mental disorder can be controlled. The outlook in patients with chronic psychoses is hopeless.

Treatment. Local therapy is unavailing. Good results may be obtained from chlorpromazine (10 to 50 mg. 3 or 4 times daily) which reduces the aggressive behavior. Sedatives are no substitutes for a thorough psychoanalytic study to determine the underlying cause.

Psychiatric therapy should be along the lines of sympathetic management, relief of tension and the correction of an unsuitable environment. Some psychiatrists get better results in dermatologic cases than others.

TRICHOTILLOMANIA

(See p. 504)

PRURITUS

GENERAL PRURITUS OF INTERNAL ORIGIN

Itching is the commonest subjective symptom of disordered metabolism. Persistent itching eventually causes secondary changes in the skin resulting from the scratch reflex. These changes consist of scratch marks, excoriations, alopecia, eczematization, lichenification and pigmentation.

Etiology. The following conditions must be considered: diabetes, liver disease, uremia, lymphoblastomas, visceral carcinoma, pregnancy, psychiatric disorders and cerebral arteriosclerosis. Cure depends upon the proper diagnosis and treatment.

Senile Pruritus

Senile pruritus is characterized by generalized itching and disturbed sleep. It occurs in patients of both sexes usually over 60

years of age. The term should be used only after a process of exclusion since systemic causes may be overlooked.

Etiology. Predisposing causes are physiologic and hormonal, resulting in decreased surface lipids, sebaceous and sweat secretions, causing a dry skin.

Differential diagnosis is from scabies, pediculosis corporis and symptomatic pruritus (diabetes, uremia, visceral carcinoma and lymphoblastoma).

Pathology consists of the usual changes found in senile degeneration of the skin.

Complications. Eczematization, localized neurodermatitis or nummular eczema may develop depending on the intensity of the itching and the individual predisposition.

Treatment. Infrequent baths followed by massage with liquid Nivea (Duke) or Lotocreme (Abbott). Usually barbiturates are necessary to control the pruritus if local medication is not effective. Resistant cases may respond to biweekly injections of estrogens or androgens and avoidance of woolen clothing, alcohol and an irritating environment. A tranquilizing drug such as meprobamate may be given 4 times daily.

Winter Itch

Winter itch (pruritus hiemalis) is a general term used to denote itching which affects certain individuals during the fall and the winter months. The trunk and the extensor and the inner surfaces of the thighs and the arms are especially involved. The symptoms are precipitated by general chilling followed by a return to warm surroundings.

Etiology includes avitaminosis A, hypothyroidism, xerosis, overdressing, sensitization to wool, strongly alkaline soaps, frequent hot baths or too infrequent bathing.

Treatment. General therapy consists of antihistaminics for their sedative effect and tonics if indicated. Locally, infrared exposures and oily lotion containing menthol or phenolated camphor are useful.

Pruritus of Pregnancy

This may be mild, moderate or severe, generalized or localized to the vulva. We have seen good results from the use of Quotane Ointment and biweekly intravenous injections of 2 to 3 cc. Benadryl.

Toxic Pruritus

This may be associated with excoriations and may result from febrile states clinical or subclinical jaundice diabetes, auto-intoxication the lymphoblastomas, and nephritis.

Diagnosis. The patient should be hospitalized and thorough laboratory and clinical studies made to determine the etiology. A biopsy of the skin to eliminate the lymphoblastomas may be rewarding.

Treatment is directed to the cause of the disorder.

Treatment of General Pruritus. Treatment depends upon discovery of the cause. Psychosomatic factors, urticaria drug eruptions, scabies, pediculosis corporis and the early stages of diabetes and lymphoblastoma should be ruled out in every case. Appropriate blood examinations should be made to exclude internal conditions.*

LOCAL THERAPY Immediate relief can be given by (1) lotions containing antipruritic drugs (menthol phenol, camphor and liquor carbonis detergens) and antihistaminics (2) starch and soda baths are extremely useful, unless the skin is very dry when $\frac{1}{4}$ 1% phenol in light mineral oil is more beneficial. Antihistaminic ointments, which are of very little value, and those containing Benzocaine may cause sensitization.

GENERAL THERAPY (1) Rest and mental hygiene (2) diet must be light—stimulants, including coffee, tea, spices, tobacco and alcohol should not be permitted (3) light clothing is advantageous (4) ultraviolet therapy in suberythema doses is soothing (5) auto-intoxication should be controlled (6) narcotics, including morphine and codeine, should be avoided since they aggravate the itching but the mild sedative drugs, reserpine, Benedryl aspirin bromides or barbiturates are generally useful.

LOCALIZED FORMS OF PRURITUS*Pruritus Ani*

Pruritus ani is a symptom complex which is limited to the anal region and results from an interplay of various factors. Sometimes it is associated with vulvar or scrotal pruritus. The anus usually presents a white sodden appearance with or without fissures or excoriations, but in some cases it appears normal. The sur-

*For more complete discussion of the subject see: Epstein, S. Ed. Panel Discussion on Allergic Pruritus, St. Paul, Bruce 1952

rounding skin may be lichenified excoriated eczematoid or pigmented depending on the duration of the disorder

Etiology There are 2 important types (1) the symptomatic, mild transient or secondary type which is associated with a known cause viz., contact dermatitis antibiotics, fissures etc., and (2) the chronic neurogenic type which persists long after the primary cause has been eliminated

The secondary or symptomatic types may be caused by one of the following (1) general diabetes uremia gout, alcoholism, hyperthyroidism and allergic states (2) reflex from immediate foci including ovarian and uterine disease bladder and prostate conditions (3) reflex from distant foci including the appendix, the gallbladder and diseases of the gastro-intestinal tract (4) drugs morphine arsenic and belladonna (5) atopic sensitization to foods or drugs in laxatives (6) eczematous contact dermatitis from sulfite toilet paper "pills" remedies or soaps (7) parasitic pediculosis pubis pinworms fungi yeasts (*Monilia*) *Streptococcus faecalis* and *Bacillus coli* (8) ingestion of antibiotics and (9) postoperative (mechanical defect in anal outlet)

The neurogenic type may be caused by (1) overtreatment, (2) fixation (often following anal surgery) and consequent scratching setting up a vicious cycle and (3) local hyperhidrosis of psychosomatic origin

In suppersensitive hypertensive and neurotic patients ordinary nonpruritic stimuli such as emotional trauma soap a bowel movement or friction from the clothing or even constipation may set up an attack of unbearable itching

Pruritus also affects more males than females The condition, if untreated eventually lead to a neurosis Hemorrhoids are found in over half of the cases but their presence may not be significant

Diagnosis The management of a case should not rest with the nonspecific diagnosis of pruritus and Every effort should be made to determine the underlying cause or causes If no local pathology can be found to explain the condition the patient should be hospitalized and a thorough examination made of the gastro-intestinal tract including the anus the rectum and the sigmoid for hidden malignancies

Prognosis. The acute types of known causation in patients under 30 respond best to proper therapy The chronic types in unstable individual may persist for many months

Treatment naturally depends upon the cause. Overtreatment should be avoided.

Symptomatic Type. (1) Simple measures should be tried first. Hydrocortone Ointment, Quotane Ointment or Gelusil containing 3 per cent Benzocaine or $\frac{1}{10}$ per cent menthol often give temporary relief (2) hot sitz baths, (3) wet packs of saturated boric acid solution or witch hazel and (4) bed rest with sedation in the severe eczematized cases. Calamine lotion should not be applied directly to the mucous membranes of the anus because the drying effect may aggravate the condition.

Soap and water cleansing should be avoided. Toilet tissue is often irritating. The areas should be cleansed after each bowel movement with a piece of absorbent cotton saturated with 1 per cent liquid phenol in Allercreme (Texas Pharmacal Co.) or Loto-creme (Abbott). If alkaline stools irritate one of the silicone ointments should be applied before the bowel movement.

The following applications may be used

Phenol	10	} for cases without visible pathology
Menthol	0.25	
Ung zinc oxide q.s	100.0	
Phenol	10	} for macerated type
Menthol	0.25	
Glycerite f tannic acid	100.0	
Castellani's solution	30.0	} for chronic fungus types
8 Paint the affected parts daily		
Silver nitrate	5.0	} for fissured types
75 wet spirits of nitre	100.0	
Hydrocortone	0.3	} for irritated type
Acid mantle cream (Dome)	30.0	
Apply three times daily		

Diet. Condiments, roughage and mineral oil should be avoided, carbohydrates limited and thorough mastication advised. Dilute hydrochloric acid (from 2 to 4 cc. in one half glass of water after meals) if tolerated tends to decrease intestinal fermentation.

Neurogenic Type The aim of therapy is to (1) prevent fixation by discussion of the cause, elimination of phobias and a complete general and proctoscopic examination (2) restoration of the perianal skin to its normal tone (3) establishment of normal bowel action and (4) control of the pruritus by local and general measures.

Sedation Antihistaminics are ineffective. Phenobarbital or one of the reserpine products reduce the intensity of the pruritus.

X-ray therapy when given by the experienced dermatologist is useful if lichenification has developed from repeated rubbing and scratching. Proctor et al. however do not favor it because of possible sequelae by unskilled operators.

Surgical and injection therapy should be avoided because the patient may be left with a permanently scarred and contracted anal ring. The Ball undercutting operation may be advisable for intractable cases.

Steroid Therapy In cases which do not respond to local and general therapy cortisone or ACTH may be given for a week or longer until the severity of the pruritic crisis has passed.

Pruritus and from antibiotics is caused by a change in the intestinal flora resulting in an overgrowth of *Candida*. Treatment consists of discontinuing the antibiotic if possible. Folic acid-iron-vitamin combinations are useful. Locally 1 to 2 per cent Hydrocortone Ointment or Mycostatin (Squibb) by mouth or in the ointment should control the pruritus.

DIRECTIONS TO THE PATIENT

Pruritus and may be cured if the following rules are observed

1. Get more rest by retiring 1 hour earlier and taking a short nap in the afternoon.
2. Relax more worry less avoid anything that upsets the peace of mind.
3. *Diet* No starchy foods, no candy or sugar (use saccharine) no condiments and no alcohol.
4. *Hygiene* Keep the parts clean. Use cotton and water for cleaning after a bowel movement. Soap and toilet paper are forbidden.
5. *Constipation* Take no pills, milk of magnesia or strong laxatives. Fruits and the mucilage type laxatives are safest.
6. *Itching Spells* Do not scratch. Use towels soaked in witch hazel or hot water (whichever gives most relief) and take a nerve pill.
7. Keep your mind off your trouble by keeping busy and avoid discussion with other people.
8. One night of scratching can undo 1 month of treatment.

Pruritus Vulvae

Pruritus vulvae is a temporary or a persistent itching of the vulva. In most cases the mucous membranes also are involved.*

Etiology includes pregnancy, menstruation, menopause, toxemias, infections, diabetes, atrophic conditions (leukoplakia and senile changes), reflex irritation from pyelitis or cystitis, infection with trichomonas, *Bacillus coli*, *Streptococcus faecalis*, Monilia, uncleanness, friction from clothing, sensitization to normal and pathologic secretions, extension of an anal dermatitis, acid urine, dribbling, sex maladjustment and perversion. The etiology must be determined if possible, in order to select the proper treatment. Fear of cancer or venereal disease or a sense of guilt may prolong the duration of simple pruritus.

Diagnosis. In children, look for threadworms. If the pruritus began at the vaginal orifice pelvic congestion may be a factor (itching worse during menstrual periods at end of day associated with fatigue, etc.) Never forget pregnancy as a cause of vulvar itching, even in early trimesters. If the itching started on the vulva check for psychosomatic states, contact dermatitis from menstrual pads, etc., urinary incontinence and vaginal discharges. A routine examination for *Candida albicans* and trichomonas should be made at the time of the first visit. In older women the labia should be carefully examined for localized neurodermatitis, lichen sclerosis et atrophicus and kraurosis vulvae.

Treatment naturally depends upon the cause.

GENERAL THERAPY. The patient should be reassured at the time of the first visit that the condition is not malignant, venereal or contagious. A thorough examination will give the patient confidence. A detailed outline of the therapeutic program is a necessity also. Special attention should be paid to the cleanliness of the affected parts. Phenobarbital and bromides are very useful in controlling the neurosis which often is associated with this condition. In hyperexcitable patients sodium amytal (0.25 to 0.5 Gm.) should be administered parenterally to control the symptoms.

LOCAL THERAPY. The antipruritic remedies, creams and lotions used in pruritus ani are useful here. Temporary relief may be obtained by using phenol 10, menthol 0.25 and lubricating jelly q s 100 or Quotane Ointment, Benedryl Cream or Hydrocortone Ointment.

*For an excellent discussion of the subject see Hunt, Elizabeth: Diseases Affecting the Vulva, ed. 4, chap. XXXII. St. Louis, Mosby 1954.

Estrogenic Therapy If senile changes are present estrogens to toleration and daily doses of 50,000 units of vitamin A may be useful.

X-ray therapy by the expert may be indicated in the chronic types if lichenification is present. However, recurrences are not unusual and resort must be made to other forms of therapy.

Infiltration Therapy In cases which do not respond to local therapy, injections of local anesthetics or alcohol may give relief for several months.

Leukotomy should be considered as the treatment of choice when leukolytic changes are evident.

Psychiatric consultation may be necessary for patients with personality disorders or deep-seated emotional conflict. The dermatologist should work with the psychiatrist to give the patient assurance and temporary relief from the pruritus.

Pruritus of the Scrotum

Itching may be mild, moderate or intense. The skin may appear normal, excoriated, eczematoid, lichenified or edematous.

Etiology The following causes should be investigated: excessive sweating, chemical dermatitis from local medication, wearing of suspenders, pediculosis pubis, secondary irritation from a contiguous tinea cruris, neurodermatitis, angioneurotic edema and diabetes (often associated with a balanitis).

Treatment Nepto Lotion (Patch), Quotane or Hydrocortone Ointment, Latocreme (Lilly) or 1 per cent phenol and $\frac{1}{10}$ per cent menthol in a greaseless cream base may give relief after the underlying cause is removed. Phenobarbital or Phenemoran may be useful for mild sedation.

Pruritus of the Scalp

There is an irresistible urge to scratch or rub the scalp producing excoriations.

Symptoms. Itching, crawling, tingling or creeping sensations occur at various times during the day but especially at night. In chronic cases the cervical or the occipital lymph nodes are enlarged. Loss of hair is infrequent unless secondary infection has occurred. The symptoms shift from one area to another.

Etiology The cause is psychosomatic and is usually evidence of frustration. The patients are usually shy, reserved, sensitive and have feelings of inferiority.

Differential Diagnosis. Pediculosis should be ruled out first. Occasionally lymphoblastoma may cause pruritus of the scalp. In acute cases, contact dermatitis from hair tonics, etc., must be considered but dermatitis is always present.

Treatment. Since local treatment is usually unsatisfactory the mild sedatives, Serpasil (Ciba) or phenobarbital should be prescribed. Some relief may be obtained from applications of 5 per cent Euresol in 60 per cent alcohol. If ineffective, psychiatric consultation is advisable.



FIG. 23. Neurodermatitis (lichen simplex chronicus) in a typical location. This condition existed for 30 years in this white male, aged 64 years. (From Dr. H. R. Cogburn.)

LOCALIZED NEURODERMATITIS

This form of eczema, also called lichen simplex chronicus, is a localized disorder characterized by one or more circumscribed patches of lichenification and extreme itching. The condition may occur as a primary disorder or a cutaneous reaction to an itching dermatitis. It is not an allergic condition.

Clinical Description The lesion is thickened, firm and pigmented or violaceous with exaggeration of the normal folds and creases. Any area constantly rubbed to relieve pruritus may be affected. The condition is common on the nape of the neck in women, the upper eyelid, the scrotum, the elbow, the knees, the inner thighs and the ankles.

Etiology In those cases in which the pruritus is caused by xerosis (dryness) the scratch-itch reflex is the determining factor. The nuchal and anorectal types are of central origin, resulting from unresolved emotional conflict. In menopausal women, estrogenic deficiency is a predisposing factor. There appears to be an individual predisposition to lichenification which results from a vicious circle of itching and persistent rubbing.



FIG. 48 A. Chronic neurodermatitis resulting from pruritus in a patient with dry skin.

Pathology Section shows hypertrophy of all the layers of the epidermis, an increase of pigment in the basal cells and cellular infiltration around the dilated vessels in the corium.

Differential Diagnosis. When the disease occurs on the elbows, the knees or the ankles, it may be confused with psoriasis, which is not an itching dermatosis. Psoriatic patches are covered with adherent silvery scales, the nails are apt to be stippled, and typical lesions are often present in the scalp and other parts of the body. The papular and the mosaic types, when they occur on the back of the neck or on the legs, may be mistaken for lichen planus. In the latter disease, however, typical violaceous, flat angular lesions will be found in scratch marks or in the vicinity of the patches. The mucous membranes may be affected

also. Lichenified types of seborrheic dermatitis usually involve the flexural surfaces. Typical lesions are present in the scalp.

Treatment. The aim of therapy is to (1) control the pruritus, (2) break up the scratch reflex, (3) prevent extension of the dermatitis, (4) heal the dermatitis and (5) solve the emotional problem to avoid recurrences.



FIG. 89 Localized neurodermatitis (lichen simplex chronicus). Essentially a chronic dermatitis, hyperkeratosis, parakeratosis, elongation of the rete pegs and spongiosis are characteristic. A moderate chronic inflammatory infiltrate is present in the cortex.

We have obtained excellent results with Hydrocortone Ointment in these cases where lichenification is not too pronounced. Although expensive it is effective when all other medication fails to resolve the dermatitis and pruritus. The scratch reflex is broken up by a discussion with the patient regarding the cause of the condition and the importance of keeping the area protected from the effects of traumatic scratching or rubbing. Pure crude coal tar liquid phenol or resin of podophyllum may be indicated in refractory lichenified types. The infiltrated areas often clear up with tar ointments which should be prescribed in mild strengths in the beginning. If the lesions are resistant to local therapy a series of x ray treatment by an expert may be necessary.

The following is an ointment which is often effective

Hydrocortone	0.5
Ung. picis liquida	5.0
Salicylic acid	3.0
Zinc oxide	5.0
Water absorbent base q	100.0

§ Appl. morning and night and keep covered with light muslin dressing.

General therapy. Emotional conflicts should be discussed, and possible solutions found. Antihistamines, barbiturates and hormones are useful in reducing the pruritus and producing a feeling of well being.

NURSING ASPECTS

The psychosomatic diseases are essentially of central nervous system origin resulting in uncontrollable itching which may be localized to certain "itch zones" or may be generalized. Since it is fear, anxiety or worry which brings most patients with skin disorder to consult the physician, an inept remark on the part of the nurse may transform a simple rash into a neurodermatitis through a mechanism called fixation.

The dermatophobias or fear complexes, consist of imaginary symptoms which usually are limited to a localized area. The basis for a burning tongue may be a fear of cancer as a result of an inappropriate remark by a medical attendant or the reading of too much cancer literature. Venereal disease is an obsession with some patients as a result of careless discussion on the part of medical attendants or close friends.

Dermatitis Factitia. This is an unusual condition consisting of a disturbed patient who produces an ulcer or a similar lesion in her skin by acid or by any means at her command in an effort to produce sympathy to gain attention or to dramatize her existence. Upon questioning she is as innocent as an angel. Suspicion of the true diagnosis dawns upon the physician as a result of study of the patient's personality the unusual or bizarre pattern of the lesion and the resistance of the lesion to proper treatment. These patients are hospitalized and, when they are out of the room "for an x-ray examination, the bed the furniture and the personal effects are searched thoroughly for the destructive agent.

Localized pruritus or chronic neurodermatitis usually affects the nape of the neck in women the anogenital area in both sexes or any part of the body accessible to the fingers. This condition is produced by a tension syndrome as a result of various environmental conflicts and is characterized by a patch of pruritic thickened skin. The patient may confide to an understanding nurse information which can be useful in understanding the motivation. Although x ray therapy and local anesthetic ointments may relieve the itching temporarily reliance must be placed upon understanding the patient's conflicts, overcoming them and channeling the tension into useful purposes.

Lichen Planus

Definition An inflammatory disease of the skin and the mucous membranes characterized by an eruption of glistering, smooth flat surfaced violaceous papules having a variable course and associated with more or less itching. The disease is discussed separately in this chapter because its exact nosologic position is unknown. It behaves like an infection and yet has psychosomatic undertones.

Clinical Description The primary lesion is a shiny smooth, flat topped angular or rhomboid papule of a violaceous color and is covered with fine adherent scales. The shiny surface is due to a stretching of the epidermis the development of the papule to the infiltration in the corium and the violaceous color to the effect of the dilated capillaries showing through the epidermis. The lesions vary from pinhead to split-pea size. Occasionally the lesions are umbilicated they may have a central horny plug. Examination of the surface of a typical lesion with a lens often will reveal a grayish streaked network (*Wickham's striae*).

The lesions may become confluent to form scaly quadrangular plaques of various sizes. However discrete lesions always are found in the vicinity of the patches. In some cases annular gyrate or linear patterns are present. An examination of the entire eruption reveals lesions in various stages of development and evolution.

Lichen planus has a selective localization which aids in the diagnosis. The following areas usually are involved flexural surfaces of the forearms and the wrist the inner thighs, the shins, the ankles the waistline the back of the neck and the penis. The occurrence of lesions in scratch marks or incisions is a diagnostic feature (Koebner phenomenon).

*For an excellent clinical discussion of the disease see Little Graham J. *Cutan Dis* 37:639 1919.

The mucous membranes are affected in about 25 per cent of the cases. The mouth is involved most frequently but most patients are unaware of it. When this is the case the eruption commonly is observed on the buccal mucosa along the lines of dentition or opposite the molar teeth. The palate, the lips, the gums and the tongue are infrequent sites. Occasionally the nails may become dystrophic or may shed.

Pigmentation which usually follows the disappearance of the lesions, may remain for several months. This is more marked in cases where arsenic or x-rays have been used. Occasionally involution may be followed by atrophy



FIG. 90. Lichen planus of the vulva. The dark area is the site of biopsy. (From Dr Robert L Howard)

Itching is a prominent but variable symptom. It is often severe in the acute cases. It may be absent entirely in the chronic types. Even though severe pruritus be present it is a remarkable fact that the patient rarely produces scratch marks or excoriations.

LICHEN PLANUS-LIKE ERUPTIONS. Occasionally an eruption simulating lichen planus may occur in pityriasis rosea and in atabrine, gold and arsenic eruptions. Sometimes it is difficult to differentiate clinically from idiopathic lichen planus.



FIG. 91. Lichen planus consisting of flat skin lacy lesions. The legs, the ankles and the mouth were involved also.

LICHENOID ERUPTIONS. These small papular eruptions may be mistaken for lichen planus because of their size, shape and color but the history, the presence of focal lesions, the absence of mucous membrane involvement and biopsy findings usually help in differentiation. Lichenoid toxic drug eruptions, *trichophyids* in-



FIG. 92 Hypertrophic lichen planus. The lesions consist of warty elevated violaceous plaques and are covered with adherent scales.

bercillids (lichen scrofulosorum) and *eczematids* are examples of lichenoid eruptions.

Types. ACUTE (GENERALIZED) LICHEN PLANUS. In some cases, the disease resembles pityriasis rosea in its early phase, but later the typical small shiny papules of lichen planus are evident in the patches. The disease runs a shorter course and the eruption is more pruritic, generalized extensive and symmetrical than the chronic variety. After from 3 to 6 weeks the eruption begins to fade. A few lesions may remain on the legs and merge into the chronic type.

CHRONIC (LOCALIZED) LICHEN PLANUS is the common variety and is characterized by a more or less generalized eruption of flat, angular glistening violaceous papules with an indefinite course.

LICHEN PLANUS ANNULARIS There are ringed lesions present, formed by a coalescence of discrete papules. The penis and the scrotum are common sites for this type.

BULLOUS LICHEN PLANUS is a rare variety which sometimes occurs on the feet in the acute types or it may appear in a generalized form following arsenic therapy. Occasionally it is confused with pemphigus vulgaris.

LICHEN PLANUS HYPERTROPHICUS (VERUCOSA) usually appears on the anterior aspect of the legs as round discoid or irregular plaques which are characterized by a chronic course a dirty gray color a warty surface and adherent scales. The surface of the lesion is covered with pits due to follicular plugging. This variety is associated with severe itching and poor peripheral circulation (See Fig 92)

LICHEN PLANUS ATROPHICUS consists of porcelain white papules or depressed white scars. Probably it is not a distinct type but the results of secondary changes in the lesions due to atrophy and a disturbance of the pigment in the basal layers.

LICHEN VITINUS consists of pink or flesh-colored shiny papules grouped or discrete usually occurring on the trunk, the flexures or the penis. Once considered a tubercled now it is believed to be a variant of lichen planus.

LINEAR OR ZOSTERIFORM LICHEN PLANUS. Occasionally lichen planus lesions especially in children may group themselves in a linear fashion like a warty nevus. In rare cases they may appear to follow along a nerve trunk. This type of lesion may be mistaken for a nevus, but the history and a biopsy decide the correct diagnosis.

LICHEN PLANOPILARIS. This variety which consists of acuminate and follicular lesions, occurs about the hair follicles. They are topped with a small horny spine resulting from a hyperkeratosis, with the formation of a horny plug in the dilated follicle. At first the lesions may be erythematous but later acquire a grayish tint. The chest the extensor surfaces and the back of the neck are frequent sites. They often are associated with typical planus papules.

LICHEN PLANUS OF THE MOUTH. The chief characteristics of lichen planus of the mouth are the opalescence and lacey border and the absence of inflammation which differentiates it from erythema multiforme lupus erythematosus, submucous thrush and mucous patches. Leukoplakia is more stationary and more sensitive to heat under the Wood's light it gives off a dull bluish-white fluorescence while lichen planus does not.

**COMPARISON OF THE CLINICAL FEATURES OF
LEUKOPLAKIA LICHEN PLANUS AND TRAUMATIC
LESIONS OF THE CHEEK MUCOSA***

	<i>(Oral) Leukoplakia</i>	<i>Lichen Planus (Oral)</i>	<i>Traumatic Lesions</i>
Sex—age	Predominantly in the male 75% of all cases. Usually develops in the older age period 40-60 years	Seen in both sexes, lightly so prevalent in the female. Age range 25 to 50 usually	Either sex, usually in young patients, particularly those of nervous temperament.
Location of the lesions	Common on tongue, palate and cheeks, beginning at oral commissure and extending back on interdental line.	Most frequently on the cheek along the interdental line in the molar region.	Usually interdental line in the molar area.
Appearance of the lesion	Irregularly shaped yellowish white leathery lesions. May be related to local sites of irritation such as rough teeth, pipe stem, etc.	On cheeks, fine interlacing lines or polygonal geometric-shaped papules. Rarely ulcerates. Purplish in color. On tongue irregularly shaped redness	Irregularly shaped grayish areas of eroded mucosa.
Surrounding mucosa	May be slight inflammatory reaction.	Usually normal.	Usually definite inflammatory reaction about the lesions. At times small submucous hemorrhages.
Subjective symptoms	Mild irritation to lips. Pain or burning sensation on eating hot or highly spiced food. Dry mouth, leathery feeling no pain on pressure unless secondary infection.	Rare — may feel rough to tongue. No pain on pressure	Pain may be marked, particularly on pressure
Result of painting with Lugol's solution	Lesions fail to take the stain	Early lesions stain normally. Older lesions stain slightly less readily than the normal mucosa.	Lesions take deeper stain than surrounding normal mucosa.
Progress of untreated lesions	Becomes thicker, fissured, ulcerated and at times undergoes carcinomatous degeneration.	May persist, regress or disappear	May become ulcerated and secondarily infected with infectious bacterial organisms.

The eruption consists of one or more delicate glistening milky white or grayish slightly infiltrated patches. The individual lesions are grouped in a mosaic, stippled lacelike annular or star like pattern. Other parts of the integument may be involved, although the disease may be limited to the mucous membranes. Subjective symptoms are unusual but cancerphobia and syphilophobia are not uncommon.

The main points in the differential diagnosis of oral leukoplakia from lichen planus and traumatic lesions of the cheek mucosa is covered in the table on page 271.

Etiology is unknown. The following theories have been held (1) that mental and emotional strain is a trigger factor but the exact relation ship is unknown. (2) lichen planus is a constitutional disease caused by a circulating toxin or virus having a specific effect on the nerve centers.

The disease is not rare. Adult from 20 to 50 years of age are predisposed. Children rarely are affected.

Present opinion regards the disease as being a cutaneous reaction in certain individuals who react in a set pattern to various drugs, fungi, bacteria or emotions.

Pathology Lichen planus has a characteristic pathology. There is a dense band of infiltration in the upper cutis which is sharply limited and consists of lymphocytes mainly. Moderate acanthosis and microscopic vesicles in the rete are usually present. The overdevelopment of these vesicles produces the bullous variety of the disease. There is also a dilatation of the capillaries in the upper and the midcorium which is responsible for the violaceous color. In the older lesions there is an increase of pigment in the basal layer. The papillae usually end in a bulbous dilatation which is often characteristic.

Pathology of oral lichen planus consists of (a) granularis, (b) acanthosis, (c) well-defined line of demarcation between the basal cell layer and the cutis, and (d) a dense bandlike infiltration in the upper corium.

Prognosis. It is impossible to determine the prognosis in advance with any degree of certainty since the course of the disease is suggestive of a psychosomatic disorder. Chronic lichen planus rarely clears up under 6 months. The factors concerned in the prognosis include the extent of the disease, the type of lesion, the reaction to treatment and general and constitutional

factors. Regardless of the type of treatment used, resolution is usually slow.

RELAPSES may take place at any time. The development of pigmentation is a hopeful sign and indicates that resolution is taking place.

Differential Diagnosis. PUNCTATE PSORIASIS may be confused with chronic lichen planus. However psoriatic lesions have a brighter red color the scales are more silvery and less adherent. Removal of the scales shows the characteristic bleeding points. In psoriasis the violaceous color, the polygonal border and the flat, shiny surface are absent. If the patient discontinues bathing for a few days, the diagnosis will become more evident.

LICHEN SPINULOSUS is an eruption of pinhead pink or skin-colored papules situated around the follicular openings, usually occurring in children. It is not familial or congenital. Frequently the abdomen the buttocks or the popliteal spaces are involved. The lesions are densely crowded into circumscribed round patches and often appear in crops. Each lesion is capped by a horny projecting spine about 1 mm. long which is formed by concentric horny lamellae. No inflammation is present but the sebaceous glands are atrophic. Treatment consists of tonics vitamin A frequent use of soap and water and local applications similar to those used in lichen planus.

LICHEN SCROFULOSORUM (See Tuberculosis Cutis Lichenoides, p. 316)

LICHEN SIMPLEX CHRONICUS (localized neurodermatitis) may resemble a patch of chronic lichen planus. In these cases it is important to look for typical lesions of lichen planus elsewhere. Histologically parakeratosis, which is present in lichenification, is usually absent in lichen planus. The cellular infiltration in the corium is more sharply limited in lichen planus.

DRUG ERUPTIONS, especially in the Negro may cause difficulty. Accentuation of the eruption following administration of the suspected drug and biopsy study should be helpful. L. H. Winer and A. J. Leeb (Lichenoid eruptions—a histopathological study. *A.M.A. Arch. Dermat. & Syph.* 70:274, 1954) discuss the distinguishing features from the standpoint of the pathologist.

LINEAR NEVI is differentiated from linear lichen planus by the history the biopsy and the course of the disease. Linear nevi are usually present since birth develop slowly and are not associated with lesions elsewhere.

LICHEN STRIATUS consists of linear lesions affects the extremities and clears up in a few weeks or months

Treatment is empirical. It is debatable whether therapy has any direct effect on the course of the disease.

ACUTE CASES should be hospitalized in order to hasten the general improvement of the patient. Phenobarbital or one of the sedative antihistaminics may be prescribed temporarily when severe itching is present. Hydrocortone or ACTH may reduce the spread of the disease and control the itching in some cases. Colloidal baths are useful in the extensive cases. Calamine lotion with phenol or camphor is a soothing local application. Ultraviolet ray baths just under an erythema dose are very useful in the generalized cases. This treatment has a soothing effect on the patient and hastens resolution. Arsenic and mercury should be withheld until the eruption enters the subacute and the chronic phases.

CHRONIC CASES X-ray radiation is not routine treatment and should be employed only by the dermatologist. It is useful in the localized and the hypertrophic types. In many cases the effect on the itching is quite rapid. Remote x ray therapy (radicular) irradiating the posterior nerve roots has proved to be disappointing.

Intercomycin may be temporarily effective in some cases but cannot be recommended as routine therapy.

Arsenic has long been used in lichen planus, but its action is at best uncertain and may aggravate some cases. It is used in the form of (1) the Asiatic pill (gr $\frac{1}{8}$ to gr $\frac{1}{32}$) or (2) a solution of potassium arsenite (gtts. 1 to 5) but is best given by injection.

Sodium arsenate	20
Phenol	20
Sterile dist. water q.s.	1000

5. Inject 1 to 5 cc., twice a week intramuscularly

The drug is given until mild arsenical symptoms occur such as puffiness of the eyes or neuritic pains when it is discontinued, and the patient given a 2 weeks rest.

Bismuth is less effective than mercury but not so painful. It may be given by mouth (Bistrimate) or by injection twice weekly Bismuth cevitaminate (C. D. Smith) or Thio-Bismol (Parke-Davis).

Mercury protoiodide (gr $\frac{1}{8}$ to $\frac{1}{4}$) or mercury with chalk (gr 1) t.i.d., prescribed with an antihistaminic, e.g. Phenergan or

Neocantergan, for several weeks often produces good results. If intestinal irritation occurs, the dose is cut down or the interval is increased.

Local applications are sometimes necessary if pruritus is present. It is advisable to prescribe a mild soothing lotion

Menthol	0.5
Glycerin	
Alcohol (70%)	
Peppermint water	ss
ad	100.0

As a rule ointments are poorly tolerated. They are contraindicated in the acute cases. In the hypertrophic types which do not respond to arsenic or x ray radiation a 40 per cent salicylic-acid plaster (Duke) 10 to 50 per cent salicylic-acid ointment, 1 to 10 per cent podophyllin ointment, carbon dioxide snow or liquid phenol may be necessary.

Mouth cases A lack of response to treatment is characteristic. The following measures may be useful (1) soothing mouth washes (e.g. Cepacol) (2) avoidance of tobacco and spicy foods (3) light cryotherapy (4) topical application of 1 per cent tincture of iodine or 20 per cent trichloroacetic acid and (5) counteract cancerphobia.

NURSING ASPECTS

Lichen planus is a localized or generalized eruption which may be caused by a virus or a blood-borne agent in susceptible individuals. The disease is neither infectious nor contagious. Facilities should be available for a complete examination of the skin and the mucous membranes. X ray therapy ultraviolet light therapy bismuth or arsenical injections and sedatives usually are employed but the disease may persist for several months.

Diseases Due to Physical Agents

CONDITIONS DUE TO FRICTION

BULLAE

PIGMENTATION

CALLUSES AND CORNS

LOCALIZED URTICARIA

CONDITIONS DUE TO TRAUMA

CONDITIONS DUE TO COLD

ERYTHEMA PERFRIO

DERMATITIS CONGELATIONIS

ESSENTIAL ACROCYANOSIS

DISEASES DUE TO HEAT

BURNS

MILIARIA AND SUDAMINA

PHOTOSENSITIZATION (LIGHT)

DERMATOSIS

POLYMORPHUS LIGHT

SENSITIVITY

ACUTE SOLAR DERMATITIS

XERODERMA PIGMENTOSUM

RADIODERMATITIS

RADIUM DERMATITIS

NURSING ASPECTS

PHYSICAL allergies to cold heat sun or mild trauma are uncommon often localized to certain exposed areas, and are probably due to the release of specific precipitable serum globulin. Treatment is unsatisfactory except for gradual desensitization measures. Many patients eventually learn their limits of tolerance to the exciting factor.

CONDITIONS DUE TO FRICTION

BULLAE

A bulla or an area of erythema may result from friction and pressure on the heels from wearing ill fitting shoes.

PIGMENTATION

Pigmentation may follow the wearing of trusses, tight hats or belts.

CALLUSES AND CORNS

Calluses and corns result from pressure or friction over a long period of time.

For a more detailed discussion of dermatoses caused by physical agents see Urbach, Erich, and Gottlieb P. M. Allergy ed. 2, chap. 17 New York, Grune & Stratton 1946.

LOCALIZED URTICARIA

Transient wheals may occur at points of pressure. One of our patients develops urticarial lesions *in situ* whenever a tourniquet is used.

CONDITIONS DUE TO TRAUMA

A large number of dermatoses occur after minor trauma some as a result of local infection (impetigo paronychia from 'picking, pyoderma, erysipelas) some as a result of anatomic defects (epidermolysis bullosa and arteriovenous aneurism) or metabolic errors (porphyria). Small repeated traumas may produce or activate malignant growths. Herpes zoster may result from trauma to the zonal segment. Granuloma pyogenicum often follows a slight injury while late syphilis of the skin often is localized at the site of trauma. A traumatic dermatitis of the scrotum from scratching is often observed in diaper rash.

CONDITIONS DUE TO COLD

ERYTHEMA PERnio

Erythema pernio (chilblain) consists of dusky red edematous areas on the ears, the nose the fingers or the toes following prolonged exposure to damp cold by individuals with a feeble or sluggish circulation. When vesiculation ulceration or gangrene occurs, the condition is called frostbite. Severe itching and burning are characteristic symptoms. The lesions are cold to the touch and often sensitive to pressure. When thawed out, extreme tenderness and itching occur in the involved areas.

Etiology The condition is more apt to affect children especially and indoor workers, the aged and those whose vitality is lowered by chronic disease. When the exposed parts are suddenly exposed to cold a contraction of the arterioles takes place with resulting vasoparesis. Sequeira emphasizes the inability of the skin to adapt itself to cold temperatures.

Differential Diagnosis. The acute and the subacute forms of lupus erythematosus may cause some confusion but the history reaction to sudden warmth and symptoms are dissimilar.

Prophylaxis. The condition can be prevented by the use of adequate clothing avoidance of exposure to cold, regular exercise and tonics to stimulate the circulation. Recurrent attacks are common.

Treatment consists of brisk friction with a mentholated ointment, warm pads, nicotinic acid amide and vitamin D₂ therapy and a ten per cent Ichthyol dressing after the circulation has been restored by friction and stimulants. Fissures should be dressed with compound tincture of benzoin.

DERMATITIS CONGELATIONIS

Dermatitis congelationis (frostbite) is a continuation of the pathology of erythema pernio with resulting tissue damage from obstruction of the circulation. Frequently the fingers, the toes, the ears and the tip of the nose are affected. In the mild cases, the disturbance is confined to a bluish-red congestion and edema, while the severe cases are characterized by vesicles, bullae, ulceration or even gangrene.

Etiology. Frostbite is the result of prolonged exposure of the wet skin to freezing temperature resulting in thrombosis of the superficial blood vessels. Predisposing factors include hyperhidrosis, exhaustion, shock and previous attacks. Frozen wet footgear is a common cause in soldiers.

Differential diagnosis is made from lupus erythematosus by the history and the appearance of the lesions and from Raynaud's disease which occurs in females, usually progresses in definite stages and is a chronic disorder.

Treatment. The emergency therapy consists of applying warmth to the affected parts until the circulation is restored, sterile dressings to prevent infection and measures to prevent ulceration of pressure points. Remedies are then applied which are used in the treatment of burns. Antibiotics to control infection, anticoagulants and blood plasma are useful when used early to prevent complications. Early active and passive exercises to stimulate the circulation is important to prevent contractures, and to minimize muscular atrophy. Conservative debridement of necrotic tissue followed by skin grafting is necessary in the advanced cases.

ESSENTIAL ACROCYANOSIS

Acrocyanosis is a vasomotor disturbance and a chronic hyperemia in children, young adults and the emotionally unstable affecting the hands and the feet which are characterized by a dusky redness and all the implications of the descriptive term "cold and clammy." The condition is normally present on the legs of young girls. Often it is associated with hyperhidrosis and

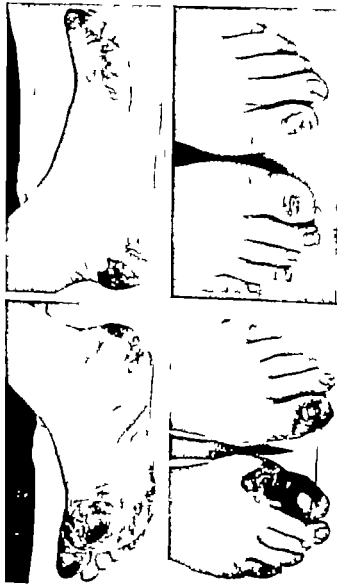


FIG. 93. Frostbite with gangrene due to vascular damage. (From Dr. E. A. Edwards)

acne vulgaris. Although the affection is not serious, a general physical examination should be made to exclude cardiac disease Raynaud's disease hypothyroidism nerve and psychic disturbances.

When the skin of a patient subject to this disorder is exposed to the heat of a radiator a hot water bottle hot paraffine dips for muscular disease or an open furnace a meshlike permanent pigmentation may occur which is called erythema ab igne.

Treatment Thyroid extract general tonics, as well as suprarenal cortical extract may be useful.

DISEASES DUE TO HEAT

BURNS

Burns may be due to dry heat steam dry ice electricity or acids. They are adequately discussed in textbooks on surgery.

MILLARIA AND SUDAMINA

Miliaria (prickly heat) and sudamina which are due to profuse sweating and poral occlusion are described on page 537.

PHOTOSENSITIZATION (LIGHT) DERMATOSES

Prolonged exposure to sunlight or other sources of ultraviolet light may produce various types of cutaneous reactions depending on the source of light length of exposure wave length of the light rays and certain hereditary factors. Little is known of the factors underlying photosensitization.*

The light dermatoses naturally occur on the exposed parts, including the face the "V" of the chest the back of the neck and the dorsum of the hands. The lesions have a tendency to disappear in the fall and reappear in the spring. This is not true however in those regions where there is abundant sunshine during the entire year.

Susceptibility to certain wavelengths of sunlight is a primary factor in the production of the following dermatoses: acute and chronic solar dermatitis, eczema solare urticaria solare nonspecific eczematous papular prurigo-like and chronic erythematous eruptions, hydrosia vacciniforme and xeroderma pigmentosum.

Sunlight as a secondary factor may initiate or aggravate lupus erythematosus pellagra berpes simplex certain drug eruptions.

For a good review of the subject as well as a study of protective measures see Kesten, B. M. and Shalkin, M. Diseases related to light sensibility. *J. A.M.A. Arch. Dermat. & Syph.* 67: 284 1953.

(sulfonamides quinine, barbiturates, arsenicals and acriflavine) dye dermatitis (erythrosin, anthracene, fluorescein, methylene blue etc.) occupational dermatitis (pitch etc.) lipstick cheilitis and pigmentation from the heavy metals (arsenic, bismuth, gold and silver) *



FIG 94 Actinic cheilitis following exposure to ultra violet sun light.

POLYMORPHOUS LIGHT SENSITIVITY (SOLAR ECZEMA)

Polymorphous light sensitivity is a chronic erythema of the exposed parts and occurs in susceptible individuals. The skin is usually eczematoid itching is marked and the eyelids tend to become edematous. After several months the skin becomes dry with atrophy and telangiectatic areas as late sequelae.

Etiology This rare dermatitis is due to a sensitization to sunlight. Most of the cases begin in the spring and persist throughout the summer until October.

Differential diagnosis is made from subacute lupus erythematosus and contact dermatitis (plants)

* For a discussion of the etiologic factors see Morgan, R. J. et al. Clinical forms of solar dermatitis, A.B.L.A. Arch. Dermat. & Syph. 67:169 1955

Treatment. Mepacrine (Atabrine) or preferably chloroquine (Aralen) are usually effective. The dose is 1 tablet t.i.d. for the first week and is gradually reduced until tolerance is established. These drugs may have a sun screening effect or a stimulating action on the pigment mechanism.* Fifteen per cent para-aminobenzoic acid in a greaseless ointment base or Neo-Afl is a useful ultraviolet light filter.

SOLAR URTICARIA

Actinic urticaria occurs during the summer months and usually affects the exposed areas. The eruption recurs each year appearing in the late spring and disappearing in the fall†.

The eruption is bilateral and consists of wheal-like papules or true wheals.

Etiology. The condition is an acquired sensitization to certain ultraviolet rays. Predisposing causes may be liver disease or intestinal toxemia. In some cases the urticaria occurs a few minutes after exposure.

Prognosis. Individual attacks disappear within a few days but recurrences are common since desensitization by gradual exposures is rarely successful.

Treatment consists of avoidance of exposure, a milk free and low protein diet, antihistaminics to reduce the pruritus and calamine or zinc starch lotions containing 1 per cent phenol or camphor.

Chloroquine may have prophylactic value.

ACUTE SOLAR DERMATITIS

Acute solar dermatitis, or erythema solare (sunburn) is a common disorder. Excessive exposure of the normal skin to the rays of the sun may provoke a dermatitis within from 4 to 6 hours. If the exposure occurs on water, ice, snow or sand, the dermatitis is more pronounced and the latent period is shortened.

Etiology. The factors involved consist of duration of exposure, thickness of the stratum corneum, intensity of the rays, and erythema threshold of the individual. The ultraviolet portion of the solar spectrum (2,800 to 3,000 Å) when absorbed by the skin results in the liberation of histamine. Children and those

For further discussion see Knox, J. M. et al. Light sensitive eruptions treated with Atabrine and chloroquine. *J. Invest. Dermat.* 22:11, 1954.

†For a discussion of the diagnostic tests see Porter, A. D. Urticaria Solare. *Brit. J. Dermat.* 56:417, 1954.

with delicate fair skins can tolerate only 50 per cent of the normal adult exposure-time. Careless exposures to ultraviolet lamps will result in the same clinical picture.

The eruption is characterized by erythema, itching and burning in the mild cases. In the more extensive cases, the eruption is vesicular, edematous or bullous and often is associated with symptoms of a toxic nature. Herpes simplex and conjunctivitis may coexist. Albuminuria is not uncommon if large areas of the integument are affected. Occasionally porphyrinuria is found in those cases where a definite sensitivity to sunlight exists.

Course The ordinary cases persist for a day or two and are followed by mild desquamation. The severe cases may last a week or longer. Pigmentation usually occurs in those gradually exposed to the natural or the artificial ultraviolet rays. Blondes and red-haired individuals, however, do not develop pigmentation. They freckle or develop a first-degree burn after exposure.

Sunburn may also be followed by (1) leucoderma (actinic interference with the enzyme system which regulates pigmentation) (2) chloasma or blotches of pigment on the exposed surfaces, (3) showers of pigmented nevi, and (4) a lowered resistance to re-exposures of intense sunshine.

Prophylaxis. Light-skinned individuals should avoid direct or prolonged exposure to the sun. Gradual exposure is the best method of preventing dermatitis. Most sunlight filters contain para-aminobenzoate or glyceryl aminobenzoate, titanium dioxide, phenyl salicylate or zinc oxide. The aminobenzoates should not be used if the patient is sensitive to Benzocaine or sulfonamides.

Treatment. For emergency purposes, cool compresses of 1 per cent Burow's solution are soothing. Phenolated neocalamine and zinc oxide lotions are useful in the acute cases. Soothing ointments are indicated in the exfoliative stage. The severe types should be treated by hospitalization, alkalies, plenty of fluids and applications of ice-cold mineral-oil compresses. Butesin picrate or Nupercaine ointment should be avoided, because of the danger of sensitization.

For the painful types the following cream may be prescribed

Benzoic acid	4.0
Boric acid	4.0
Liquid petroleum	12.0
White petroleum jelly	64.0
Anhydrous lanolin q.s. ad	100.0

The following lotion is beneficial in the acute cases

Neosalamine	
Zinc oxide	℥i
Tannic acid	6.0
Glycerin	1.5
Rose water	q.s. ad
	100.0

FRECKLES

(See p. 455)

HYDRA VACCINIFORME

(See p. 152)

XERODERMA PIGMENTOSUM

Xeroderma pigmentosum a precancerosis is a rare congenital and hereditary form of light sensitivity due to certain specific invisible rays of the light spectrum. The condition begins in infancy as a photophobia when the child is first exposed to light. In the beginning before the characteristic degenerative changes set in the skin is dry, rough and scaly. Soon this is followed by dark freckles which later become black in color. As the disease progresses white flat atrophic areas, dilated capillaries and warty growths make their appearance. When the condition is fairly well advanced it simulates the characteristics of chronic radio-dermatitis. Warty growths or keratoses appear from time to time on the skin and the exposed mucous membranes and are of grave concern because they usually degenerate into squamous-cell carcinoma. Ectropion of the lower lids is commonly present as a result of keratoses on the cheeks which gives the patient a squint. Porphyrin studies should be made to determine the role of this sensitizer in the disease.

Pathology. The disease passes through the following stages over a period of several years: (1) inflammatory, (2) hyperpigmentation, (3) atrophic and (4) malignant degeneration. The pathology is suggestive of changes seen in senile skin and roentgen dermatitis. The malignant growth is a squamous-cell or basal-cell one, but melanomata and sarcomata are not uncommon.

Etiology. Consanguinity in the parents has a bearing on the transmission of the disease which is a recessive characteristic and affects both sexes. The exciting cause is exposure to light containing wavelengths approaching those of x-rays and the absence of a physiologic protective mechanism.

Course. The disease runs a chronic course, but life is short for these unfortunates. Squamous-cell carcinoma eventually develops in some of the keratoses and results in death from metastatic involvement.

Differential Diagnosis. Multiple lentigenes appear later in life and are not associated with atrophy or telangiectasis. In chronic arsenical pigmentation the keratoses, if present are not imbedded in an atrophic skin with telangiectasis, and a history of taking the drug can usually be obtained.

Treatment. Keratoses should be destroyed with the cautery or by electrodesiccation as soon as they form. Exposure to direct sunlight should be avoided. Cortisone therapy may improve the irritability of the skin and reduce photophobia (Guzman). Veterinary red petroleum affords some measure of protection.

Only by early surgical intervention and frequent observation can these unfortunates be spared an early death.*

RADIODERMATITIS

X ray Reactions. The use of x ray therapy in diseases of the skin may produce transient effects many of which, while of minor importance should be understood by the physician. A temporary or a permanent dryness may result from the inhibition of sebaceous-gland and sweat-gland secretions. Temporary epilation may occur from 16 to 21 days following suberythema doses. Regrowth usually takes place after 2 or 3 weeks. Irritation of the skin with redness following rubbing or slapping is a warning sign that erythema is about to develop. Hyperpigmentation is a frequent result of x ray therapy. The occurrence of freckles is usually of a temporary nature and is seen in dark-skinned individuals. Tanning also may occur in those with dark skins and is apt to be permanent. Depigmentation following suberythema doses is not uncommon in Negroes.

ACUTE RADIODERMATITIS. After a latent period of several days between the exposure and the first visible signs of dermatitis, various changes occur in the skin. The extent of the dermatitis may be classified as follows:

First Degree Burn. There is itching and burning and a well-defined erythema over the exposed area which is followed in 2

*For discussion of the disease from the surgeon's viewpoint, see Brackerton, W. S. and Fosterthwait, R. W. "Xeroderma pigmentosum," *Ann. Surgeon* 18:130-57 1951.

The following lotion is beneficial in the acute cases

Neosalamine	
Zinc oxide	41
Tannic acid	6.0
Glycerin	1.5
Rose water	6.0
qs. ad	100.0

IRICKLES

(See p 455)

HYDROA VACCINIIFORME

(See p 152)

XERODERMA PIGMENTOSUM

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Etiology Consanguinity in the parents has a bearing on the transmission of the disease which is a recessive characteristic and affects both sexes. The exciting cause is exposure to light containing wavelengths approaching those of x-rays and the absence of a physiologic protective mechanism.

men including radiologists, are also frequent victims. Most of the cases occur on the hands and the fingers. The early symptoms consist of itching and burning of the affected parts, especially during cold weather. The skin becomes dry, the hairs drop out, and fissures form over the joints and the folds of the skin. Wrinkles, thin atrophic spots, pigmented macules and dilated capillaries appear throughout the affected areas. Even the nails share in the degenerative process and become opaque, ridged, brittle, fissured atrophic and pigmented.

If keratoses occur they should be excised and examined microscopically because of the danger of malignant degeneration.

Etiology. Eller and Eller (*Tumors of the Skin*, ed. 2, p. 265, Philadelphia, Lea & Febiger) state that the susceptibility of certain individuals to radiodermatitis is debatable. It is a fact, however, that children, the aged, blondes and redheads are more sensitive to radiation. Radiodermatitis may be caused by overdosage (in accurate records, mechanical errors, persistence in treating an unyielding lesion), carelessness and ignorance. One fourth of the cases result from therapeutic radiation for malignancy of internal organs, thyroid disorders, etc. 10 per cent result from repeated fluoroscopic examinations, and about 10 per cent of the cases are the result of irradiation for hypertrichosis.

Atomic radiation, experimental or in wartime, may cause severe burns.

Pathology. The extent of the pathology depends upon the degree of the reaction. In typical burns of the second degree there is atrophy of the epidermis, degeneration of the connective tissues, dilatation of the blood and the lymph vessels and degeneration of the vascular walls.

Differential Diagnosis. Depending on the stage and the location of the dermatitis, the ulceration or the alopecia, various atrophic conditions must be considered. A biopsy is usually necessary.

Treatment. THE ACUTE CASES are treated according to the principles employed in the therapy of burns. Wet dressings and emollients are useful in the early stages to allay the itching and the burning.

THE CHRONIC CASES without ulceration should be treated with soothing oils or ointments. Alvagel (General and Marine Lab.) and Hydrosulfosol Ointment (Lient) when used for several months may retard degenerative changes. If no improvement occurs after 1

year plastic surgery should be considered. Keratoses should be destroyed by electrodesiccation as soon as they appear.

ULCERS. Sometimes small areas are healed with the juice of the *Aloe vera* leaf which can be obtained from most botanical gardens. The leaf is split lengthwise and the cut edge is strapped to the ulcer. Relief from pain is sometimes remarkable and healing is rapid and progressive in many cases.

If the ulcer is extensive and complicated by fibrous changes, the standard treatment consists of immediate wide excision followed by a plastic operation, using full-thickness skin grafts. Involvement of cartilage requires plastic surgery also.

RADIUM DERMATITIS

Physicians and technicians who handle radium tubes, plaques or emanations may be subject to acute or chronic radiodermatitis similar to that caused by the x rays. The fingers and the hands usually are affected.

Treatment is similar to that used in x ray dermatitis.

NURSING ASPECTS

Proper protection and prophylaxis can prevent many accidents caused by ultraviolet light or x ray radiation. If the nurse is entrusted with the care of the radium supply special precautions are necessary. The danger from burns caused by hot water bottles, diathermy apparatus and electrical equipment should be apparent at all times.

Ultraviolet light Burns. The nurse should be acquainted with the erythema dose of the machine and with the idiosyncrasies of thin-skinned and blonde and red-haired patients. To avoid reactions, the first dose never should be more than one minute. A time clock in good working order is a necessity as well as a tape measure and black sheets to cover the areas where treatment is not desired. Distraction and absent-mindedness must not be permitted. The machine must be cleaned daily and any defects should be reported at once to the physician. If an erythema dose is given the patient should be advised beforehand and a lotion should be prescribed otherwise a phone call from the patient is inevitable.

X ray Burns. If the nurse operates the x ray machine she must be fully trained in the following: preparation of the patient, protective measures, administration of proper dosage, self-protec-

tion and record-keeping. All vital parts should be shielded. The uterus in pregnant women and in women of childbearing age always should be protected from radiation. Relatives should be kept out of the treatment room because of the danger of scattered radiation. Any unusual reaction from a previous treatment or any fears mentioned by the patient should be reported to the physician.

Radium Therapy Radium never should be handled except with proper protective gloves or forceps. At the end of the working day all radium plaques, needles and tubes should be counted, examined and placed in their lead containers under lock and key. Supplies of aluminum foil, rubber cement and filters should be available. Radium must never be left unexposed on a table or a shelf. Dressings used on radium-treated patients must not be discarded until the nurse is sure they are radium free. The length of exposure must be noted carefully.

Radium technicians should have a physical examination and blood counts every 6 months. Frequent inspection of radium containers to detect leakage is advisable. From the standpoint of protection against loss, accurate records should be kept of the amount and the location of all radium containers.

The Deep Infective Dermatoses

DIPHTHERIA OF THE SKIN
 ANTHRAX
 CHANCROID
 GRANULOMA INGUINALE

LEPROSY
 TULAREMIA
 KERATODERMIA BLENNORR
 HAGIUM
 NURSING ASPECTS

The diagnosis of this group of diseases rests mainly with the laboratory. Except for chancroid the majority of them are rare in this country. Unless the examiner has a high index of suspicion, the diagnosis of anthrax, tularemia and leprosy may be delayed. The newer drugs have improved the prognosis of this group considerably.

DIPHTHERIA OF THE SKIN

Diphtheria of the skin is a rare infection that may occur from accidental contact with the virulent organisms of the disease. Painful ulcers with a black eschar, impetiginous and eczematoid lesions may result. Complications include peripheral neuritis and myocarditis. Toxic symptoms depend on the extent of the skin involvement.

Etiology. (1) Direct primary inoculation through an abrasion or a banal inflammation of the skin or (2) secondary infection of a pre-existing dermatosis with the *Corynebacterium diphtheriae*. Minor epidemics may occur in the poorly nourished, the unhygienic and those living in crowded quarters.

Diagnosis depends first on a high index of suspicion, then positive cultures and animal virulence tests. Prompt amelioration of the pain following the administration of diphtheria antitoxin is suggestive.

Differential Diagnosis. Facilitious ulcers, ulcers caused by anaerobic bacteria and trophic ulcers must be considered.

Treatment. As soon as the diagnosis is suspected and while waiting for the report on the culture, the patient should be iso-

lated, and an intramuscular injection of 50,000 units of diphtheria antitoxin should be administered. Also it is advantageous to dress the lesions with daily applications of the antitoxin or compresses of penicillin solution (500 units per cc.) The patient should not be discharged until repeated cultures from the skin as well as from the mouth, the nose and the throat, are negative for the diphtheria bacillus. Bed rest is important until activity is arrested to avoid cardiac complications.

ANTHRAX

Anthrax (malignant pustule) is a rare infection of the skin with the anthrax bacillus and is accompanied by symptoms



FIG 97 Anthrax Not black crust (Dr Harold Ellington case)

of a grave constitutional character. The disease usually ends fatally unless treated early. Depending on the portal of entry three varieties are recognized: the cutaneous, the gastro-intestinal (rare in man) and the pneumonic.

The cutaneous type is acquired by exposure to infected animal material containing *Bacillus anthracis* which enters the tissues as a result of a break in the skin. There are three main characteristics which are pathognomonic: (1) a carbunculoid lesion with central sloughing surrounded by an area of brawny edema; (2) a localized suppurative adenitis; and (3) a septic temperature.

A few days after inoculation a pustule appears on the site of infection and becomes surrounded by an edematous inflammatory zone. This soon alarms the patient. As the pustule enlarges in size it breaks down with the formation of a black necrotic slough. The inflammatory area in the immediate vicinity is a very red, deeply infiltrated and covered with tense vesicles. A purulent bloody discharge escapes from the floor of the ulcer and tends to increase in amount with the extension of the infection. If the disease progresses, metastatic abscesses from the septicemia appear over the entire body, the temperature curve assumes a septic character, and the constitutional symptoms become aggravated. Death occurs after from 3 to 10 days from septicaemia, septic bronchopneumonia, nephritis or meningitis.

Etiology. The exciting cause is *Bacillus anthracis*, a large rod-shaped gram positive bacillus which occurs singly or in pairs in smears. The organism forms spores and grows in chains on artificial media. The disease occurs sporadically in those whose occupational contacts consist of working with wool, hides or animal bristles (cattlemen, woolsorters and butchers). Improperly sterilized shaving brushes may harbor the organism.

Anthrax occurs endemically in three fairly well-defined areas in the United States: (1) southeastern North Dakota and northeastern Nebraska; (2) the Delta regions of the lower Mississippi Valley; and (3) a belt along the Texas Gulf Coast. Sporadic cases occur in cities where felt, wool and bristle industries are located. It is not the serious health problem of former years (only 42 human cases were reported in 1952) although animal cases are fairly common.

Diagnosis is made from the clinical appearance of the lesion, the history and the occupation of the patient, the presence of the

organisms in the smears or the blood stream and by keeping the possibility in mind

Differential diagnosis should be made from carbuncle.

Prognosis. Best results can be expected in those cases treated with combined serum and penicillin. The gastro-intestinal types are usually fatal because often diagnosis is made in the late stages.

Treatment. Extreme precautions should be taken by the medical and the nursing staff to prevent self infection from the discharges. As soon as the diagnosis is made, antianthrax serum (from 50 to 150 cc.) if available should be administered for its specific effect on the course of the disease. If not available, large doses of sulfadiazine and penicillin or chloramphenicol and blood transfusions are indicated. Excision of the primary lesion not only is unnecessary but manipulation may destroy the local barriers and render septicemia a more likely complication.*

Prophylaxis. The incidence can be reduced by (1) warning labels on material imported from countries where the disease is endemic, (2) personal protective measures on the part of employees (3) education and (4) facilities for early diagnosis and therapy

CHANCROID

Chancroid (ulcus molle) is an ulcerative condition of the genitals caused by the *Hemophilus ducreyi* and usually is characterized by secondary inflammation of the inguinal glands (bubo)

Etiology. The disease is transmitted through sexual intercourse. It is about ten times more frequent in the Negro race. Males are affected more often than females, but the latter are frequently carriers.

Clinical Description. The primary lesion begins as a small ulceration which develops 2 or 3 days after sexual exposure. The characteristic lesion is a painful ulcer with sloping edges and a dirty-grayish floor. Most of the cases in males occur on the preputial orifice, the internal surface of the prepuce or the frenum, rarely on the perineum, the scrotum or the anus. In the female the labiae are common sites. Extragenital lesions on the lips and the tongue are rare. In about 50 per cent of the cases the disease is complicated by inguinal adenitis which may be uni-

* Gold, H. and Boger, W. P. Newer antibiotics in the therapy of anthrax, New England J. Med. 244:391 1951

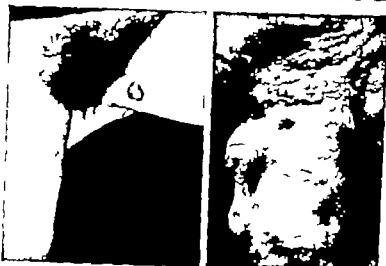


FIG. 98 (Left) Chancroid, or soft chancre. The lesion started as a vesicopustule 3 days after exposure and soon developed into a non-indurated ulcer.

FIG. 99 (Right) Granuloma inguinale. Fibrosis, ulceration and lymph edema—frequent complications. A foul odor is present.

lateral or bilateral. A superimposed fusospirochetosis or syphilitic infection is not uncommon.

Diagnosis is made by direct smear or from cultures on fresh rabbit's blood media. However the organisms may be absent in half the cases if the ulcer has been treated previously. Diagnosis also is verified by a specific controlled intradermal test consisting of the injection of 0.1 cc. of Ducrey vaccine (Lederle). After 48 hours an infiltrated red papule appears which persists for several days. A darkfield examination for *Treponema pallidum* should be made at the same time as mixed infections are not uncommon.

Pathology. The lesion advances from papule to abscess to ulcer, all stages showing evidence of an acute inflammatory process. At first the epidermis is edematous but soon it becomes the site of numerous epidermic abscesses. The subepidermal zone of the corium contains a dense infiltrate of polymorphonuclear cells, chains of Ducrey bacilli and dilated lymphatics and capillaries. Later abscess formation gives way to ulceration.

Differential diagnosis. The disease must be differentiated from syphilis, lymphogranuloma venereum and granuloma inguinale.

Treatment. Inguinal adenitis is treated by evacuation of the pus and instillation of from 1 to 1.5 cc. of 7 per cent tincture of iodine.

SULFADIAZINE is the drug of first choice except in sulfonamide sensitive or sulfonamide-resistant cases. One tablet 4 times daily for 2 weeks is usually curative. The drug does not suppress a superimposed syphilitic infection which is an advantage over antibiotics.

AUREOMYCIN in doses of 250 mg. every 4 hours for 3 days, if tolerated is the most effective antibiotic.*

CIRCUMCISION should be avoided until three months after complete healing. Dorsal-split incision may be necessary in the acute cases if marked phimosis interferes with drainage.

LOCAL APPLICATIONS must be selected with care. If the ulcer is moist and discharging only mild antiseptic wet dressings should be used otherwise there is danger of obstructing drainage and promoting an inguinal adenitis.

GRANULOMA INGUINALE

Granuloma inguinale is a chronic, indolent, deep ulceration of the genitals and the surrounding areas characterized by slow extension and the formation of dense fibrous scars. Most cases occur in Negroes in the Southern states and in the larger cities of the country. The incubation period varies from 2 to 12 weeks.

The disease begins on the penis or the labia majora as a hard nodule which breaks down. By slowly undermining the surrounding tissues, it forms a dirty foul painless ulcer. When the condition has existed for several months, the floor of the ulcer becomes covered with velvety red papillomatous fungoid masses. As the ulceration advances contiguous parts are involved, with extension to the vagina or the anal region in the female and to the thighs, the buttocks, the perineum and the scrotum in the male. The lymph nodes are not involved unless secondary infection is present.

Varieties. Nodular, scirrhous, deep ulcerative, hypertrophic and cicatricial types.

*For details of administration see Robinson, R. C. V. Newer antibiotics in the treatment of venereal diseases, *Am. J. Syph.* 34:273, 1939.

Etiology The infection belongs to the group of venereal diseases. Most of the cases are acquired through sexual intercourse. It appears to be established that *Donovanian granulomatis* a short plump bacillus, is the specific cause.

Pathology The epidermis is ulcerated and pseudo-epitheliomatous hypertrophy is a constant feature. The massive cellular infiltrate in the corium consists of leukocytes, plasma cells and histiocytes some of which contain Donovan bodies.

Diagnosis. The long duration and the slow course the absence of a positive V D R L or Kahn test the finding of Donovan bodies in the tissue and the absence of lymphatic involvement are important facts in making a diagnosis. Smears are unsatisfactory in advanced cases because of the presence of numerous secondary organisms.

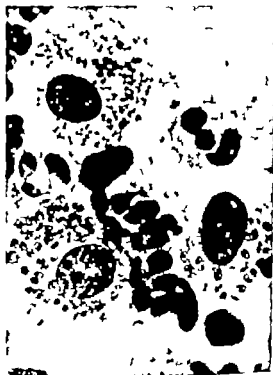


FIG. 100. Donovan bodies in a tissue section of granuloma inguinale. (From Dr. R. O. Noofa)

The slow extension of the ulceration is characteristic. If scrapings are negative the cut edge of the biopsy section should be used for making the smear which is stained with the Wright stain. The Kohnblith Intracutaneous test is also of value.

Differential Diagnosis. The condition must be differentiated from late syphilis, chancreoid actinomycosis adenocarcinoma, amebic ulceration and hydradenitis suppurativa by repeated biopsies, smears and cultures.

Course. Spontaneous cure is rare. Healing results in dense fibrous replacement, leaving marked destruction of the tissues. Recurrences are common unless treatment is continued after apparent cure.

Treatment. Streptomycin (1 Gm. every 6 hours for 5 days) will cure the average case. Aureomycin and Chloromycetin are effective in streptomycin-resistant cases.*

LEPROSY

Leprosy is a chronic systemic polymorphous disease found mostly in tropical and subtropical climates. It is caused by the *Mycobacterium leprae*. The infection is characterized by involvement of various tissues of the body especially the skin, the mucous membranes and the nerves, and often a variable course. Fear of this disease is based mainly on Biblical references. In countries where the disease is prevalent no more fear of leprosy exists than of syphilis in the United States. The disease occurs in two forms the infectious nodular and the benign maculo-anesthetic, although nerve involvement is common to both types. After a variable period the nodular eventually passes into the anesthetic type.†

Incubation period probably is between 3 and 10 years, the longest of any of the infectious diseases.

The onset is very gradual with intermittent fever loss of weight and appetite. The disease consists of three stages (1) the quiescent, or stage of dissemination (2) the stage of activity (3) the stage of degeneration which is characterized by a breaking down of the tissues.

* See Greenblatt, R. B. et al. Five year study of antibiotics in the treatment of granuloma inguinale. *Ann J Syph* 36 186, 1952.

† A detailed discussion of the subject will be found in Strmons, R. D. G. Ed. Leprosy, chap 19 in *Handbook of Tropical Dermatology* vol. 1 Houston, Elsevier 1952.



FIG. 101 (Left) Cutaneous leprosy with involvement of the corneas, resulting in total blindness.

FIG. 102 (Right) Maculo-anesthetic leprosy. This type is not infectious.

Cutaneous Types. THE *LEPRO* REACTION occurs during the invasive period and is characterized by an eruption of refractory urticarial or erythema multiformelike lesions. These eventually develop into the nodular type.

LEPROMATOUS LEPROSY. The macular lesions of the early type change from a light-pink to a brown color and gradually develop into oily or shiny nodular lesions. The supra-orbital regions become thickened and assume a characteristic appearance. Nodular lesions may appear on the nose, the ears, the cheeks, the back of the hands, the trunk and the extensor surfaces of the extremities. The vocal cords, the nasal mucosa and the cornea also become involved eventually in the leprosy process. When the nodular lesions ulcerate they become infectious.

In this type the lesions are unstable. If *leprae* are found in the lesions and the lepromin reaction is negative in 90 per cent.

Although the scalp hair is profuse, an alopecia limited to the outer third of the eyebrows is an important sign.

TUBERCULOID TYPE. The first sign of nerve involvement is the presence of one or more hypopigmented or hyperpigmented, anesthetic or hyperesthetic patches. The color of the lesions is usually light pink or brownish. They vary considerably from

dime size to the size of the palm of the hand or larger. The involved nerves include the ulnar, the peroneal, the sciatic, the median, the radial, etc. When the ulnar nerve is affected atrophy of the interosseous muscles results in the "leper claw" which is another characteristic feature. Involvement of the nerves may result eventually in extensive destruction of an extremity with dry gangrene or paralysis. In this type *Al leprae* are absent, the lepromin test is positive and the lesions are stable.

Indeterminate and borderline types are recognized also.

Etiology The exciting cause is the acid-fast *Mycobacterium leprae*. However the organism never has been cultivated or inoculated into man or laboratory animals. For this reason an antitoxin or a serum never has been discovered.

Leprosy exists the world over but is endemic only in those areas characterized by a high rainfall and a hot climate.

The disease probably is transmitted by contact but the conditions that make infection possible are not understood at this time.

Leprosy is primarily a disease of filthy and unsanitary methods of living. The cockroach and the bedbug may harbor the infective organism. The higher the scale of civilization the lower the incidence of leprosy in the community. Close contact is essential in the dissemination of the disease, but apparently leprosy is no more contagious than tuberculosis. "Where there is one leper there is bound to be another" is an old axiom which, while true in the endemic countries, apparently does not hold in the United States, where sporadic cases occur from time to time.

Recent investigations tend to disprove the theory that the disease is hereditary. However most cases are acquired in early childhood. In the United States foci of the disease are present in the Gulf Coast states and among the Mexicans and the Orientals of the West Coast.

Pathology Leprosy is an infective granuloma. Naturally the pathology varies according to the stage of the disease. The main feature is an infiltration in the corium and the subcutaneous tissues consisting of connective tissue plasma cells and mast cells. The characteristic lepra cells should be looked for in the infiltrate. These are large vacuolated cells containing one or more nuclei and clumps of lepra bacilli.

Sections of nerve tissue removed for diagnostic purposes contain nodules in the connective tissue sheaths. Nerve degeneration is present, depending on the degree of pressure atrophy. Leprosy

sarcoid lesions do not contain bacilli because of the allergic reaction in the tissues which destroys the organisms.

Diagnosis is made by observing (1) a thickening of the supra-orbital ridges (2) a nodular eruption on the ears (3) palpable postauricular and ulnar nerves (4) the presence of bizarre brownish infiltrated patches on the trunk, (5) areas of altered sensation (6) ulcerations (7) muscular atrophy (8) paralyses, (9) contractures of fingers or toes (10) alopecia of eyebrows, and (11) inhibition of sweating in localized area. The pilomotor nicotine test is used to detect peripheral nerve degeneration.

The A. D. R. L. test is usually negative in the uncomplicated cases although it may be positive in the advanced cases. A positive serology also may indicate that the patient has a cross-infection with yaws.

Microscopic Diagnosis. The organisms must be demonstrated in the nasal discharges or in the suspected tissues. If nasal smears are negative iodides should be given for several days to promote the secretions. The serum in blister lesions, produced with solid carbon dioxide should be examined for *M. leprae* if other tests are negative.

A piece of the suspected tissue should be excised or a 20-gauge needle inserted and material aspirated for bacteriologic investigation. No anesthetic is required because the tissues are already anesthetic.

Differential Diagnosis. The macular types rarely are confused with leukoderma or toxic erythemas. The nodular types may resemble late syphilis, yaws, sarcoid or neurofibromatosis, and the neural types may be confused with Raynaud's disease, arteriosclerotic gangrene or syringomyelia.

Prognosis has improved considerably since the advent of the sulfones and the antibiotics. Recovery may occur in both forms of the disease after a long period of treatment. The nodular type is more resistant to therapy. In untreated cases, the general health of the patient is affected sooner or later by tuberculosis, amyloid nephrosis or some other chronic debilitating disease. A positive lepromin reaction (Mitsuda Rost test) indicates a high resistance to the infection.

Prophylaxis. All infectious cases should be reported to the proper authorities. They also should be isolated and segregated. All contacts especially members of the immediate family should be examined every 6 months for a period of 5 years. Infants and

children of leprous parents should be reared separately by non-leprous relatives preferably

Treatment. In the United States active cases are quarantined in the Federal Carville Leprosarium, where they are surrounded by cheerful and hygienic surroundings and supplied with a nourishing and high-caloric diet. If they are able to work, occupational therapy is prescribed to overcome depression and pessimism. The lepra reaction (acute symptoms) often responds to cortisone or ACTH. Chaulmoogra oil and its ethyl esters (Chaulmoestrol) have been replaced by sulfones. Streptomycin, vitamin D₂, Promizole and Diasone are effective in clearing up the lesions and reducing complications. In resistant types and in cases that do not respond to the sulfones, isonicotinic acid hydrazides appear promising. The dose is 3 to 5 mg. per Kg. body weight in equal divided doses every 12 hours for several months. Toxic effects from these drugs are common, so that frequent blood counts and clinical observation are necessary.

Criteria of Cure. Patients are considered cured if (1) no new lesions appear for 3 years, (2) the lepromin test is positive (3) no bacilli are present in nasal smears, skin or mucous membranes and (4) the potassium-iodide test is negative.

TULAREMIA

Tularemia is a chronic infectious disease caused by the *Pasteurella tularensis* and characterized by cutaneous, glandular or systemic involvement.*

The three general types of the disease are (1) the primary cutaneous (ulceroglandular) (2) the primary ophthalmic (oculoglandular) and (3) the cryptogenic (typhoid type).

PRIMARY CUTANEOUS TYPE. After an incubation period of from 3 to 7 days, an infiltrated papule or nodule which may have been preceded by a scratch or abrasion appears on the site of inoculation (finger or thumb). At the onset the patient usually complains of malaise, chills or a fever which ranges from 100° to 103° F. The primary lesion may persist as a punched-out ulcer, a diffuse crusted swelling or a sporothrixlike nodule. The lesion rapidly becomes painful and swollen, followed shortly by a nodular lymphangitis (rosarylike) and a painful enlargement of the

* A more complete discussion will be found in Meyer, K. F. *Tularemia bacterium tularensis*, pp. 476-483 in *The Pasteurella*, chap. 19, in Dubos, R. J. Ed. *Bacterial and Mycotic Infections of Man*, ed. 2 Philadelphia, Lippincott.

Diagnosis is made by the clinical appearance of the lesions, the presence of a focus of chronic gonorrhea and of typical gonorrheal arthritis limited to one or more joints.

Course is chronic with exacerbations and remissions.

Etiology Its production appears to be allergic in nature. The rarity of the condition considering the immense number of gonorrheal cases can be ascribed to a state of anergy which exists in ordinary specific urethritis.



FIG. 101. Keratoderma blennorrhagicum in a 21-year-old male. Associated lesions were a polyarthritis, a chronic posterior urethritis and a prostaticitis. The patient ran a low-grade fever and lost 15 lbs. at the syndrome appeared.

Pathology The essential feature of the lesion is a parakeratosis not a hyperkeratosis. The disease consists of an inflammatory process in the epidermis and the edematous papillae with the formation of vesicles filled with serous exudate which eventually form crusts.

Differential Diagnosis Histologic sections of old lesions of keratoderma blennorrhagicum may simulate psoriasis if the vesic

For a discussion of modern theories in relation to the causative factors, see Ladd, E. and Hughes, J. D. Etiologic considerations of keratoderma blennorrhagica, Arch. Dermat. & Syph. 54:150, 1946.

ular stage has disappeared and if the infiltrate has been absorbed. The crusts of keratoderma blennorrhagicum differ from the scales of psoriasis in that the former may be removed easily in toto, while the flaky scales of the latter disease are removed with difficulty.

Reiter's syndrome consists of low-grade fever, conjunctivitis, arthritis, an acute nonspecific urethritis, followed by skin lesions resembling those of gonorrheal keratoderma. Most of the cases have occurred in young white males. From 6 to 12 weeks are required for resolution.

Treatment. In most of the mild cases treatment directed toward the gonorrheal complications produces a rapid effect on the skin manifestations. Fever therapy, sulfadiazine and foreign-protein injections are useful. General measures to increase the patient's resistance also are indicated, including large doses of vitamin A. Penicillin or Aureomycin may be used in those cases which do not respond to sulfonamides, but the response is variable. ACTH and hydrocortisone are useful in the acute stages with painful arthritis.*

NURSING ASPECTS

The deep infective dermatoses are all potentially infectious, especially among laboratory personnel. Nurses working in wards where patients with diphtheria of the skin are being treated should have a Schick test. Rubber gloves are a necessity in applying dressings to patients with anthrax, chancreid, granuloma inguinale, leprosy, or tularemia. All discarded dressings should be burned. Patients receiving sulfonamides for chancreid or sulfones for leprosy should be observed at frequent intervals for drug eruptions and blood counts should be made weekly to detect anemia or neutropenia. Nurses handling streptomycin should be aware of the possibility of contracting contact dermatitis of the hands from the drug.

*See Myerson, R. M. and Katzenstein, L. Gonorrheal keratosis successfully treated with corticotropin, *J A M A* 149:132, 1952.

Tuberculosis of the Skin

LOCALIZED (INOCULATION)

FORMS

PRIMARY TUBERCULOSIS

COMPLEX

TUBERCULOSIS VERRUCOSA

CUTIS

TUBERCULOSIS CUTIS

COLLIQUATIVA

(SCROFULODERMA)

TUBERCULOSIS CUTIS

ORIFICIALIS

LUPUS VULGARIS

HEMATOGENOUS TYPES

TUBERCULOSIS CUTIS

MILIARIS ACTUTA

TUBERCULOSIS CUTIS

LICHENOIDES

TUBERCULOSIS CUTIS

PAPULONECROTICA

LUPUS MILLIARIS DISSEMI

NATUS FACIE

ROSACEALIKE TUBERCULID

(LEWANDOWSKY)

ERYTHEMA INDURATUM

SARCOID REACTION

SARCOIDOSIS

NURSING ASPECTS

The cutaneous reaction to the presence of the tubercle bacillus is polymorphous depending on the presence or the absence of an internal focus of tuberculosis the presence or the absence of allergy to the tubercle bacillus geographic considerations, the race and the age of the patient the virulence and the number of organisms, the route of infection and all those composite factors that are called the soil. Most lesions are characterized by a typical tuberculous histology. Tubercle bacilli may be present in some varieties and absent in others. In some cases the tuberculin reaction is positive in others it is negative.

Usually the portal of entry is the deciding factor that determines the type of clinical lesion. Scrofuloderma results from direct infection of the skin from underlying tuberculous lymph nodes or sinuses tuberculosis verrucosa cutis from direct inoculation through a break in the skin tuberculous ulcers of the tongue by secondary contact with sputum containing viable organisms erythema induratum and millary tuberculosis of the

skin by the hematogenous route from an active visceral focus.

The majority of patients with cutaneous tuberculosis belong to the lower social stratum. Naturally this means low resistance, poor sanitation and hygiene and a high incidence of childhood infections.

Most of the cases are caused by the human type of tubercle bacillus, a few are caused by the bovine type. The avian form rarely causes skin tuberculosis.

The most practical classification of these conditions is based on the prognosis.

Diagnosis should not depend on clinical signs alone. biopsy, Ziehl-Neelsen stain for the tubercle bacillus, animal inoculation and tuberculin tests are important for correct analysis of a case.

The localized types usually are associated with an absence of sensitivity to the tubercle bacillus (anergy) while the hematogenous forms occur in individuals whose skin has been sensitized to an internal focus by dissemination of the infection.

The prognosis of cutaneous tuberculosis has become more favorable with the advent of streptomycin and isonicotinic acid.

Chemotherapy of tuberculosis of the skin. In addition to good general hygiene and nutrition, drug therapy is necessary to stimulate the defensive mechanism against the tubercle bacillus and to effect a bacteriostatic or bactericidal action on the organism. The type of agent used depends on the type of disease, its availability and the presence of complications that might increase toxicity. Drug resistance is common, intolerance frequent and relapses after insufficient dosage must be guarded against. In general a combination of drugs has an added therapeutic effect and smaller doses may be used.*

CALCIPIROL. This is the safest drug and although it has no known bacteriostatic or bactericidal effect, it has a specific action on the tissues in which the bacilli are present. The dose is 150,000 I U daily for 3 to 6 months. Contraindications are senility, hypertension, renal disease and active pulmonary infection. The drug should be given with a bactericidal agent (dihydrostreptomycin or isonicotinic hydrazide).

*For a recent discussion of the subject, see: Proceedings of the Tenth International Congress of Dermatology pp. 193-245, Brit. Med. Assn. London, 1952.

ISONICOTINIC ACID HYDRAZIDE The dose is 3 to 5 mg. per kg. body weight or about 50 mg. 4 times daily. The drug if tolerated should be continued for at least 2 or 3 months.

P-AMINOSALICYLIC ACID (PAS) has an inhibitory effect on the growth of the tubercle bacillus. Usually it is given in combination with dihydrostreptomycin. The dose is 5 to 15 Gm. daily for 30 to 60 days.

DIHYDROSTREPTOMYCIN is given in doses of 0.5 to 1 Gm. daily in divided doses for about 6 weeks if tolerated. Drug resistance and toxicity may force reliance on other agents.

THIOSEMICARBAZONE may be effective in cases with mucosal involvement. The dose is 150 to 300 mg. daily. Drug reactions are not uncommon.

GOLD INJECTIONS are now obsolete because of the frequency of toxic reactions and unfavorable results.

LOCALIZED (INOCULATION) FORMS

PRIMARY TUBERCULOSIS COMPLEX

Primary tuberculosis complex is a form of true inoculation tuberculosis or the so-called tuberculous chancre. This type occurs in individuals who previously have been free of a tuberculous infection. Therefore it affects children and is rare in adults. The usual lesion is an indurated indolent deep-seated sharply circumscribed, crusted ulcer surrounded by a zone of erythema. It is analogous to the Ghon primary lung tubercle.

Regional adenitis or lymphangitis practically always is present. If the glandular enlargement is marked, a "cold abscess" or ulceration may result. Most of the cases have been reported on the face, the extremities and the genitals. The chin is a favorite site.

Etiology An abrasion or a scratch is necessary for infection to develop. Contact with sputum containing viable bacilli must take place.

Pathology Tubercle bacilli are present in the lesions and in the draining lymph nodes. The histology is that of true tuberculosis with typical tubercle formation and marked caseation necrosis. The tuberculin test is positive in low dilutions only.

Diagnosis is made by the history of a "pimple" which becomes ulcerated after a week or two followed after a few weeks by regional adenopathy. Biopsy is confirmatory.*

*For a more detailed discussion, see Miller F. K. W. Recognition of primary tuberculosis infection of the skin and mucous membrane. *Lancet* 115, 1953.



FIG. 104 (*Left*) Tuberculosis cutis verrucosa, the result of external inoculation in a highly resistant individual.

FIG. 105 (*Right*) Tuberculosis colliquativa (scrofuloderma) overlying tuberculous cervical lymph nodes.

Treatment is seldom necessary the lesion often healing spontaneously but resistant types may require general ultraviolet light baths, high-vitamin diet, calciferol or isonicotinic acid and general tonic measures.

TUBERCULOSIS VERRUCOSA CUTIS

Tuberculosis verrucosa cutis is a warty type of inoculation tuberculosis in patients with good resistance. The lesions may be ulcerated, granulomatous, warty or crusted. In its early stages the disease may resemble a common wart. As a rule, the condition consists of a patch of brownish-red circumscribed dime-sized to palm-sized lesions, covered with fine warty vegetations or thin crusts. A violaceous areola usually surrounds the lesion. A small amount of pus can be expressed from the surface in the pustular types. When spontaneous healing occurs the infiltrated area is replaced by a smooth pliable scar. Most of the cases occur on the fingers or the dorsum of the hands from which the disease may extend around to the palms or upward to the wrists.

Etiology The disease is acquired from contact with tuberculous cadavers, infected animals or tuberculous sputum. A lesion similar in pathologic structure has been reported to have occurred in swimming pools. The term "anatomic wart" is applied to the type of localized tuberculosis which occurs in physicians or attendants at autopsies, or meat handlers or inspectors.

Pathology is similar to that of lupus vulgaris. Tubercle bacilli are numerous in the lesions. The tubercles are chiefly in the papillary layer of the skin and consist of lymphocytes, plasma cells, large mononuclear cells and a few giant cells of the Langhans type.

Prognosis is good.

Diagnosis is made by biopsy a history of a previous tuberculous infection the clinical picture and in doubtful cases, guinea-pig inoculation. Some cases occur in patients with a positive sputum. The tuberculin test is positive only in low dilutions.

Differential diagnosis is from blastomycosis, verrucous lupus vulgaris, verruca vulgaris, loderma bromoderma and vegetating pyoderma.

Treatment Excision is the ideal treatment for small lesions. At the same time sections can be made for study. The cautery and electrodecaction are methods of second choice. Streptomycin (1 Gm. daily for 6 weeks) or isonicotinic acid (50 mg. t.i.d. for 5 to 15 weeks) is usually effective in the more extensive cases.

TUBERCULOSIS CUTIS COLLIQUATIVA (*Scrofuloderma*)

This is a common form of secondary involvement of the skin resulting from an underlying tuberculosis of the lymph nodes, the joints, or the bones. The cervical the mandibular and the preauricular regions usually are affected.

The condition is characterized at first by firm dusky red subcutaneous nodules. These gradually enlarge and become adherent to the overlying skin by means of a plastic exudate. The resulting interference with nutrition and the pressure atrophy cause a rupture of the surface structures, with the production of purplish undermined ulcers which exude a serosanguinous purulent secretion.

Palpation of the lesion reveals a hard or a doughy marble-sized to lemon sized chain of matted infected lymph nodes. The overlying skin is undermined and perforated by numerous sinuses which produce the characteristic linear or oval ulcers. After healing occurs, fibrous tissue replaces the ulcerative areas with characteristic cordlike nodular or cribiform scars.

Etiology The disease is common in children who suffer from glandular joint or bone tuberculosis. Unpasteurized milk is the probable source of infection.

Pathology The subcutis and the deeper parts of the corium are affected primarily with abscess formation, tubercles, the presence of tubercle bacilli and more or less infiltrate, consisting of lymphocytes and plasma cells. The edges of the ulcers show pseudo-epitheliomatous proliferation.

Course. This form of tuberculosis runs a more rapid course than the other localized types. However it has the distinction of being more amenable to treatment. The disease continues for several months, or even years, resulting eventually in spontaneous healing.

Differential diagnosis is from pyogenic lymphadenitis, late syphilis, actinomycosis, sporotrichosis and early Hodgkin's disease.

Treatment. Streptomycin (except in cases with hypertrophic scarring) or calciferol therapy is worth a trial before deciding on total excision of the infected gland. *p*-Aminosalicylic acid or isonicotinic acid may be useful in streptomycin-resistant cases. The underlying bone or joint disease should receive the attention of an orthopedic surgeon and the general health should be cared for by a pediatrician. Local applications should be avoided.

TUBERCULOSIS CUTIS ORIFICIALIS

These ulcers are the result of direct implantation of the tubercle bacillus into mucocutaneous surfaces or mucous membranes from an active focus in the lungs, the intestines or the genitourinary organs. The lesions, which ulcerate from the beginning consist of painful, oval, shallow ulcers. The lip the tongue or the buccal mucosa are favorite sites in patients with laryngeal tuberculosis the anal region in cases of intestinal tuberculosis and the genital region in cases of tuberculosis of the bladder. The tuberculin test is usually negative. Young adults with lowered resistance are predisposed.

Pathology The lesion consists of a large number of typical tubercles which lie in the deep corium. Tubercle bacilli are numerous in the early cases. A characteristic of the disease is the rapid caseation of the tubercles, with subsequent ulceration of the overlying mucosal surfaces.

Diagnosis is made by the presence of a primary focus, positive smears from the ulcer and biopsy study.

Differential diagnosis must exclude mucous patches, lupus erythematosus, periphigus and Vincent's fusospirochetosis.

Prognosis. Since this type of tuberculosis occurs in the late stages of the disease the prognosis is grave.

Treatment. The best method of destroying the lesions is electrocoagulation. The original focus must receive suitable attention. Streptomycin or isonicotinic acid is useful in the debilitated if not contraindicated.



FIG. 106 Tuberculosis cutis orificialis. (From Dr Beatrice H. Kahn)

LUPUS VULGARIS

This disease also called tuberculosis cutis luposa is a chronic type of inoculation tuberculosis which is characterized by very slow growth more or less extensive ulceration and the presence of typical apple-jelly nodules in the patches. The condition begins in childhood or early adolescence as a pinhead-sized deep-seated nodule. When several of these nodules coalesce a dusky red violaceous patch is formed which spreads very slowly by peripheral extension. If the patch is compressed by a glass slide, several firm discrete dull-red semitranslucent nodules from 1 to 2 mm in diameter are visible in the deeper parts of the lesion. These apple-jelly nodules (so named from their color) represent a collection of microscopic miliary tubercles in the dermis. They also have a tendency to appear in the scars of healed areas. This is an important observation which helps to distinguish the disease from late syphilis. Extensive involvement often results in hideous disfigurement.

Three varieties of the disease occur (1) smooth, or atrophic, (2) hypertrophic and (3) verrucous, depending on the state of

activity. The disease involves the head in 95 per cent of the cases, especially the nose, the upper lip, the cheeks, the ears and the neck. Old patches are subject to squamous-cell degeneration, especially if previously treated with arsenic or x-ray radiation.

Etiology. The disease begins in childhood or adolescence, rarely in adult life. Most of the cases occur in the lower social



FIG. 107. The hypertrophic type (*lupus vulgaris*) (of 30 years duration) with gradual extension and areas of ulceration. (From Dr. John C. Slaughter, Jr.)

classes of the temperate zone. The disease may represent a superinfection, i.e. an exogenous infection in a patient with a pre-existing visceral tuberculosis.

Infection may follow trivial trauma, with direct inoculation of the tubercle bacillus. Occasionally the disease starts from a broken-down tuberculous gland or joint. About 40 per cent

of the cases are due to infection with the bovine type of tubercle bacillus. A chest expert should examine the patient for evidence of visceral tuberculosis.

Pathology shows a wedge-shaped infiltration in the corium with the characteristic epithelioid tubercles, consisting of epithelioid cells, small round cells and plasma cells. A few Langhans' giant cells are also present. Caseation necrosis is not marked. Practically always tubercle bacilli are present in the sections.

Diagnosis. The presence of apple-jelly nodules activity and healing slow progress and biopsy studies are usually sufficient evidence. Where the diagnosis is in doubt, animal inoculation is carried out.

Course The disease is chronic, with periods of comparative quiescence and activity throughout life. Nodules in old scars may flare up at any time. When the disease affects the cheeks, the lower eyelids are apt to be involved eventually with a resulting ectropion. If the nasal cartilages are destroyed, the nose assumes a beaklike appearance.

Differential Diagnosis. *LUPUS ERYTHEMATOSUS* begins in later life and is of shorter duration. Stippling is present, imperfect scarring and apple-jelly nodules are absent.

BASAL-CELL CARCINOMA has a rolled border no apple-jelly nodules are present and the pathology is characteristic.

LATE SYPHILIS (gumma) consists of larger nodules, arranged in a circinate or serpiginous pattern. There is a history of shorter duration the Kolmer test is usually positive and the bony rather than the cartilaginous structures are apt to be invaded. *Lupus vulgaris* tends to involve the tip while syphilis involves the bridge of the nose. The nodules of late syphilis do not develop in the scars.

MEDICAL TREATMENT No permanent cure can be effected without general hygienic measures, maintenance of nutrition and good environmental conditions.

Isoscotinic acid hydrazide (Rimifon, Marsilid, Hydrazid) is perhaps the most effective drug. It may be alternated with vitamin D₂ (calciferol).

Dihydrostreptomycin is used in the vitamin D₂-resistant cases (1 Gm. daily) or in combination with PAS or isoscotinic acid hydrazide. In refractory types the antibiotic may be injected directly into the lesion (250 mg. in 2 per cent procaine solution)

PHYSIOTHERAPY The Finsen light formerly was used in European medical centers for the treatment for the vitamin D₂-resistant lesions. General ultraviolet-light baths are indispensable in building up the patient's resistance. Caustery or electrodesiccation techniques are useful but scarring is inevitable.

LOCAL THERAPY is useful in selected cases. Promin ointment (5 per cent) may be tried in the ulcerative types.

SURGICAL TREATMENT (avascular lesions not reached by drug therapy) consists of curetting the lesion and packing the space with powdered potassium permanganate which results in a slough. Small lesions are best excised by extending the incision to one quarter of an inch from the diseased area and cutting down to the fascia.

Resistant nodules should be fulgerated and the wound dressed with 15 per cent PAS ointment.

The criteria of cure is the absence of "apple-jelly" nodules for 5 years without relapses.

HEMATOGENOUS TYPES

If the patient has a focus of infection in the lungs, the lymph nodes, the kidneys, or the bones, the skin is apt to become allergic to the tubercle bacillus, its mutations or toxins. A breaking down of the diseased tissue usually results in a dissemination of the bacteria in the blood stream and eventually they reach the allergic skin. This produces a cutaneous reaction which takes place in certain areas where the concentration of the organisms is greatest. When the lesions are small and appear in showers or crops in a hyperergic skin they are called tuberculids. Large annular plaques or infiltrated areas in a hypoergic or an anergic skin are known by the general term of "sarcoid." The clinical picture in both tuberculid and sarcoid eruptions is always subject to change with the immunologic state and the character of the soil. With the exception of acute miliary tuberculosis, none of the hematogenous types is serious as regards life expectancy.

Diagnostic criteria for tuberculids (1) a primary focus is present but not always apparent (2) the lesions involute when the primary focus heals (3) the skin is allergic to tuberculin in dilutions of 1:100,000 or over (4) no tubercle bacilli can be found in the lesions (5) the onset is sudden as a rule and (6) there is symmetrical distribution. At present the pathogenesis of tuberculids is unsettled.

TUBERCULOSIS CUTIS MILIARIS ACUTA

Postexanthematic milary tuberculosis is essentially a disease of childhood. Many of the cases follow measles or whooping cough. The eruption which is symmetrical and usually appears suddenly consists of numerous indolent brown h-red acuminate follicular papules. These resemble a small papular acne. After a short time a few of the lesions degenerate into round or oval, sharply defined ulcers, surrounded by a violaceous border. Occasionally some of the ulcers contain a few milary nodules. Section reveals a characteristic pathology and numerous tubercle bacilli in the lesions. If the chest plate is taken a hilus-gland tuberculosis usually is found.

Prognosis is poor. Death usually results from tuberculous meningitis.

Treatment is systemic. Streptomycin combined with isonicotinic acid hydrazide is worth a trial.

TUBERCULOSIS CUTIS LICHENOIDES

Also called lichen scrofulosorum, this type consists of grouped coin-sized patches of indolent round or acuminate papules, which appear about the hair follicles on the flanks and on the front of the trunk. These characteristics give the lesions a goose-skin appearance. They are usually the color of normal skin or reddish brown and have a small scale or spine at the summit. After several weeks the eruption may disappear without leaving a trace or small scars may be left behind. Itching is usually absent.

Etiology. The disease usually occurs in children or adolescents and represents a high degree of allergy. The eruption persists if a tuberculosis focus is present, and tends to disappear when the internal infection is cured.

Differential Diagnosis. The condition must be differentiated from lichen planus which consists of an eruption of flat-topped shiny angular lesions, affecting the flexural surfaces of the wrists, the arms and the neck; from lichenoid syphilis, in which there are other signs of syphilis and a positive Kolmer; from papular contact eczema, by the history and the absence of itching; and from toxic eruptions in children which are usually fleeting.

Diagnosis is made by the history, the presence of a visceral focus, the presence of positive von Pirquet and tuberculin tests and by biopsy studies.

Pathology In well-developed cases, numerous discrete tubercles are found in the upper third of the corium, surrounded by a zone of lymphocytes and young connective-tissue cells. These usually are arranged about the follicles and sweat glands. Caseation is rarely found.

Prognosis is usually favorable. The disease persists so long as the focus of tuberculosis is present.

Treatment. Cod-liver oil should be administered internally and also used in the form of a 5 per cent ointment for local application. Rest and a high-calorie diet also are indicated.

PAPULONECROTIC TUBERCULIDS

This type consists of small papules with central necrosis, which come in crops and leave pigmented depressed varioliform scars. The characteristic lesions are purplish painless and persist for several weeks. The eruption, which is usually symmetrical is often unnoticed. In most of the cases the face the ear lobes, the extensor surfaces of the arms and the forearms, the backs of the hands, the buttocks, the legs and the feet are affected. The disease requires from 2 to 8 weeks to run its course.

Etiology Most of the cases occur in young adults who suffer from acrocyanosis or some other form of skin tuberculosis. The eruption tends to become worse in the winter. An internal focus of tuberculosis is usually present.

Diagnosis is made by the clinical characteristics, the reaction to tuberculin in high dilutions and the characteristic pathology.

Differential diagnosis is from acneiform drug eruptions, acne vulgaris, and staphylococcal pyoderma. Upon removing the crust, a sharply defined central area of necrosis is found in the center of a papule in papulonecrotic tuberculids. This distinguishes the eruption from acne vulgaris.

Prognosis. In some cases there is eventually spontaneous cure, but the eruption usually persists, with remissions and exacerbations so long as the visceral focus exists.

Pathology Tubercle formation with caseation necrosis usually is found in the middle and the upper corium, which is surrounded by a nonspecific inflammatory reaction. When the necrotic nodules break through the epidermis, small crusted ulcers are formed.

Treatment consists of attention to the general health, fresh air high-vitamin diet, general ultraviolet radiation and the ad-

ministration of cod-liver oil and iron tonics. Vitamin D₂ (calciferol) isonicotinic acid hydrazide and streptomycin are also effective in most cases but scarring is inevitable.

Antibiotic ointments may be used locally to retard secondary infection.

LUPUS MILIARIS DISSEMINATUS FACIE

This is a follicular type of hematogenous tuberculosis of the skin. The eruption which is usually symmetrical and affects the face, the cheeks and the neck, consists of grouped dusky-red or golden yellow tiny papules. They are apt to be infiltrated and often contain a few apple-jelly nodules. After retrogression, punched-out scars are left at the site of the old lesions. In some cases a phlyctenular conjunctivitis is present.

Etiology. The affection is limited to young adults. A focus of visceral tuberculosis, which renders the skin anergic to tuberculin, is usually present.

Differential diagnosis is from early syphilis, *ioderma*, *acne vulgaris* and the rosacealike tuberculid.

Pathology. The lesions are centered about the blood vessels. They have features of both *lupus vulgaris* and papulonecrotic tuberculids. Tubercle bacilli sometimes are found in the sections.

Course. The eruption tends to disappear spontaneously.

Treatment. Isonicotinic acid or PAS combined with streptomycin are usually effective.

ROSACEALIKE TUBERCULID (*Lewandowsky*)

This condition consists of an eruption of small infiltrated papules on a hyperemic background. The eruption is limited to women and usually occurs on the forehead, the cheeks and the ears sparing the nose. The individual lesions are yellowish brown, smooth, flat or crusted. If the hyperemia is marked the lesions may be masked so that close inspection is necessary to make a diagnosis. The skin is often hyperallergic to tuberculin in high dilutions.

Etiology. A family history of tuberculosis or frank pulmonary tuberculosis is usually present.

Diagnosis, which often is decided on insufficient evidence is made by the biopsy and the clinical appearance of the eruption.

Differential diagnosis is made from *acne vulgaris*, *rosacea*, follicular seborrheid and telangiectatic lupus erythematosus.

Prognosis. The disease clears up spontaneously after several months.

Pathology consists of circumscribed tubercles in the upper or the midcorium, often near a sebaceous gland. Laymon and Schoch (Micropapular tuberculid and rosacea, Arch. Dermat. & Syph. 58.286, 1948) found a similar tuberculoid pathology in some cases of true rosacea and advise caution in making the diagnosis.

Treatment consists of the use of streptomycin vitamin D₂ or isonicotinic acid which is preferred for proven cases. General tonics and rest are important.

ERYTHEMA INDURATUM

Tuberculosis indurativa (Bazin's disease) is a chronic, deep nodular and ulcerative bilateral disease affecting the calves of the legs in the presence of local circulatory disorders.

Before the eruption appears the nodules can be felt, lying deep along the venules of the subcutaneous tissues. After several days or weeks, the lesions become more evident when the overlying skin turns a purplish red. The characteristic eruption consists of discrete painless inflammatory nodules and irregular ulcers. The ulcers heal spontaneously after several months, leaving punched-out pigmented scars. However new lesions appear from time to time as the old ones are absorbed or break down.

The eruption localizes on the backs of the legs especially the lower third of that region. In practically all cases the disease is bilateral.

Etiology Erythema induratum affects girls and young women almost exclusively. Most of the cases occur during the winter months. There is often a history of pulmonary or glandular tuberculosis. The disease is probably due to the toxins of the tubercle bacillus.

Pathology The dense infiltration is perivascular and is situated in the lower third of the corium and the subcutaneous tissue in the early stages. As the disease progresses the infiltration extends upward to the epidermis, resulting in ulceration.

Diagnosis is made by the presence of a symmetrical eruption of dusky painless, soft nodules on the lower third of the calves of the legs, usually in women, eventual ulceration with scarring, the presence of a visceral focus of tuberculosis and the charac

teristic pathology with tubercles in the corium and the chronic course. When the pathology is doubtful guinea-pig inoculation with the suspected tissue is advisable.

Differential Diagnosis. IN LATE SYPHILIS (gumma) the patient is usually older, the Wassermann is positive, the lesions are asymmetrical and is not confined to the calves of the legs.

IN ERYTHEMA NODOSUM the lesions are of shorter duration and go through the "bruise cycle." They are more inflammatory, are tender and involve the anterior aspect of the legs. The nodules never break down in erythema nodosum.

VARICOSE ULCERS usually occur in older patients. They are surrounded by an area of lymphedema, are usually single rather than multiple and are associated with the presence of varicose veins.

DERMATITIS NODULARIS NECROTICA is a type of erythema induratum in which evidence of tuberculosis is absent.

Prognosis. This disease is refractory to treatment. New lesions may continue to form as the old lesions are absorbed. This may continue for several years.

Treatment. The usual tuberculosis regimen is indicated. Ulcerated cases should be treated by absolute bed rest and the use of fixed bandages (elastoplast) similar to those used in varicose veins, to restore the circulation. General ultraviolet radiation is useful as a resistance-builder. Streptomycin when given with IAS or calciferol is valuable in the refractory cases. Cod-liver oil ointment should be used locally.

SARCOID REACTION

Sarcoid are benign solitary or multiple cutaneous or subcutaneous nodules or plaques which represent a cutaneous reaction to various internal and external stimuli. These lesions are not related to sarcoma or to sarcoidosis.

Etiology. External causes are implantation of glass, talc, sand, hairs, asbestos or beryllium which may be detected by examining the biopsy specimen under polarized light. Internal causes are infections including syphilis, leprosy and also drug reactions.

Course. Those cases resulting from infections usually disappear spontaneously.

Differential Diagnosis. Tuberculids, lichen planus and early lesions of lupus erythematosus and sarcoidosis.



FIG. 108. Sarcoid (Boeck) of the plaque type. (Dr. Kendall Frost's case)

Pathology Sarcoid consists of naked epithelioid "tubercles" in the upper corium, with a minimum of surrounding cellular infiltration and an absence of caseation. Foreign bodies may be found in the tubercles."

Treatment. The foreign body type should be excised. The therapy of the infectious type is that of the causative disease.

SARCOIDOSIS

This is a chronic systemic infection of unknown etiology which may affect any structure in the body especially the skin the bones the lymph nodes and the lungs. Extensive pathologic changes may occur with a minimum of constitutional symptoms.

Clinical Description. CUTANEOUS LESIONS. Sarcoids consist of localized and generalized eruptions. The lesions have the features of a chronic infectious granuloma. The cutaneous manifestations include lichenoid, nodular plaque papular annular diffuse erythematous and scaly macular types.

Three characteristics of sarcoid are (1) its multiformity and (2) its tendency to gradual progression with frequent remissions and relapses and (3) absence of ulceration. Spontaneous healing may occur without evidence of atrophy.

The usual sites affected are chest back legs face and neck although any part of the body may be involved. In Negroes the

ears the eyelids the nares, the back of the neck, the upper chest and the buttocks often are affected. The cutaneous eruption in this race is frequently of the keloidal nodular type. The lesions vary from the size of a split pea to that of a silver dollar. The color is usually brownish red bluish red or yellowish depending on the stage of the disease. Generalized adenopathy is often present.



FIG. 109. Boeck's sarcoid (From Dr. Beatrice H. Kuh.)

VISCERAL LESIONS. The visceral lesions consist of granulomatous infiltrations in the lymph nodes, the lungs and other viscera and cystic changes in the long and the short bones. The disseminated variety (sarcoidosis) has been regarded as being a gener

alized reticulo-endotheliosis or a benign lymphogranulomatosis. Skin lesions are rare in this type (34 per cent). Lungs, bones, glands, tonsils, parotid glands, palate, larynx, nasal mucosa, liver, spleen, eyes and ears may be a part of the general invasive proc-

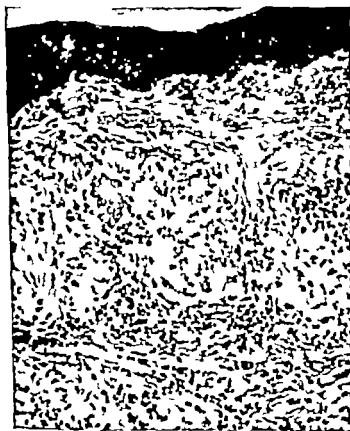


FIG. 110 Section from ear lobe in a case of sarcoidosis, showing large tubercle, giant cell and the scanty infiltrate.

ess. When the lungs are affected a peculiar reticulation, or marbling occurs resembling military tuberculosis. Lesions of the bones include a fusiform spina ventralislike swelling of the fingers and the toes, and cystic and honeycomb lesions of the phalanges, the long and the short bones (osteitis cysticum multiplex). Dyspnea, fever

fatigue and weight loss often occur in the pulmonary types, especially in Negroes. Cachexia develops in cases of long standing.

Etiology The skin lesions may represent the sites of a rapid destruction of tubercle bacilli by the local immunity in the tissues in a highly sensitized individual. The eruption occurs during the anergic phase of the infection.

The disease usually begins insidiously in early life. It is twice as common in the Southern states as in other parts of this country. In Negroes the condition is apt to be atypical and complicated by lesions of true tuberculosis. Various theories have been brought forward to explain this type of infection including the following:

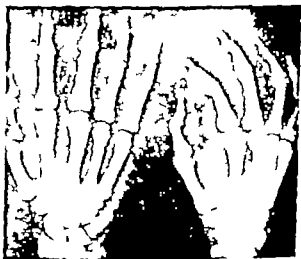


FIG. 111 Sarcoidosis involving metacarpal bones.
(Dr. W. W. Ham)

(1) the original focus is pulmonary (2) filtrable virus or unrecognized bacillus (3) a form of tuberculosis in a highly resistant subject (4) a modified form of leprosy occurring in the temperate zone (5) a benign form of lymphogranulomatosis (6) the activation of a hematogenous tuberculous infection by trauma and (7) a nonspecific response to tuberculous toxins or lipoids.*

The factors in favor of a tuberculous etiology are the tuberculouslike histology, the not infrequent association of the disease

*For an excellent review of the subject, see Curtis, A. C., and Grell, R. H. Sarcoidosis. A review. *31 Clin. North America* 33:310-324, 1949.

with other forms of tuberculosis of the skin and the occasional presence of visceral tuberculosis, especially in Negroes. The exact etiology is still under investigation. Michelson doubts the tuberculous factor because of the favorable effect of the corticosteroids in many cases.

Against this view are the absence of the organisms in the tissues, negative animal-inoculation and tuberculin tests and failure of the lesions to flare up following tuberculin injections.



FIG 112 Pulmonary sarcoidosis. (Dr W W Hunt)

Pathology The typical histology in the white patient consists of compact masses of circumscribed naked epitheloid tubercles. Caseation necrosis does not occur in true sarcoid. The lymphocytic infiltration about the tuberculoid reaction is scanty but Langhans giant cells are usually present. Asteroid bodies (non specific) and Schaumann's inclusion bodies often are found in the infiltrates.

The tuberculin reaction is usually negative because of the presence of anergy. However the Negro who is subject to an atypical form of skin tuberculosis is hyperergic to tuberculin. The albumin-globulin ratio usually is disturbed in the systemic types. No acid-fast organisms are present in the tubercles.

Differential Diagnosis. The disease is difficult to diagnose unless one has a wide knowledge of dermatology and internal medicine. Sarcoidlike reactions may occur at one time or another in the following diseases: leprosy, syphilis, histoplasmosis, trichophytosis, lymphoblastoma and foreign-body granuloma. The skin lesions may be confused with those of late syphilis, lupus vulgaris, leprosy and true tuberculosis.

Prognosis is good unless visceral disease is present. A change in the tuberculin reaction from negative to positive usually indicates the development of true tuberculosis, a serious prognosis and a change in the immunologic pattern.

Course. The disease is subject to frequent remissions and exacerbations. Death is usually due to cardiac failure* but a fatal termination from tuberculosis is rare except in Negroes.

Diagnosis is made by biopsy of the skin lesions, abnormal albumin-globulin ratio (70 per cent), the anergy to tuberculin, the Kveim reaction (see p. 19), sternal bone-marrow studies, absence of disturbance of the general health, the presence of visceral pathology, negative animal-inoculation tests and the absence of tubercle bacilli in the tissues.

Treatment. The principles of treatment include the general care necessary in tuberculosis, the building up of the resistance and therapy directed to the local lesions.

Vitamin D₂ (calciferol) and the corticosteroids (if there is no evidence of latent tuberculosis) have definite value in the disseminated types but do not appear to affect the skin lesions.

NURSING ASPECTS

The localized types of tuberculosis of the skin may be potentially infectious to the nurse but ordinary protective measures are sufficient to guard against this possibility. When dressing tuberculous ulcers of the mouth or the anogenital areas, gloves should be worn. Discarded dressings should be burned. The tuberculids and sarcoidosis are not infectious. Patients with all

*See, Gill, I. A clinical study of the causes of death in sarcoidosis, *Acta dermat. venerol.* 34: 47, 1954.

forms of tuberculosis require the same general treatment, diet and care given to those with the pulmonary form. Nurses should be aware of the possible reactions from streptomycin and calciferol therapy. Those handling streptomycin should be familiar with the signs of contact dermatitis of the hands.

Syphilis

SEROLOGIC TESTS FOR SYPHILIS	PRENATAL SYPHILIS
PATHOLOGY	TREATMENT OF PRENATAL SYPHILIS
ACQUIRED SYPHILIS	SYPHILIS IN PREGNANCY
PRIMARY SYPHILIS	TREATMENT
SECONDARY SYPHILIS	SERORESISTANT SYPHILIS
TREATMENT OF EARLY SYPHILIS	TREATMENT RESISTANT SYPHILIS
LATENT SYPHILIS	ANTIBIOTICS
LATE SYPHILIS	PUBLIC HEALTH ASPECTS
TREATMENT OF LATE CUTANEOUS SYPHILIS	NURSING ASPECTS

SYPHILIS is a chronic constitutional disease involving all the tissues of the body. It is caused by the *Treponema pallidum* (*Spirochaeta pallida*). The disease becomes generalized within a few hours after inoculation. It is characterized by frequent relapses during which the patient may become infectious to others. If untreated it progresses through three stages (primary secondary and tertiary) which represent either the life cycle of the parasites or peculiarities in the mechanism of the resistance of the host.

The penicillin era has reduced the incidence of the disease considerably so that cases with skin manifestations are seldom seen at present. However unrecognized and undertreated cases may present visceral lesions that may be serious.

Sources of Infection. THE INNOCENT CASES are acquired through legal marital relations kissing and contact with infectious material including drinking glasses towels, dishes, musical instruments, lipstick, pipes, surgical and dental instruments and transfusions.

The VENEREAL CASES are acquired by sexual contact with an infected partner especially the "party girl" young inexperienced prostitutes or low-class professionals

Incidence varies according to race age sex, social and economic status and environment. According to the Federal Security Agency of the U S Public Health Service New England states report the smallest number of cases. The South Atlantic states, the largest number. The death rate from the disease is highest among the nonwhites in the South.

Difficulties of control have been inadequate treatment of our migratory population the high incidence (20 per cent) in the rural Negro population lack of investigation of contacts due to *insufficient state funds, and juvenile delinquency*

Characteristics of the *Treponema pallidum*. The morphology of the organism can be studied best in its living state in the darkfield. It is a spiral, slender refractile, moderately motile anaerobic parasite having corkscrew forward and backward and slightly undulating movements. The number of spirals ranges from 6 to 24. It is practically impossible to grow it on artificial media.

The viability of the organism is such that drying immediately kills it. Soap is the cheapest and most effective spirochetal germicide. When the organism is present in moist secretions it may remain viable for as long as 11½ hours, but its virulence probably disappears long before. The organisms die under blood-bank conditions within 72 hours. In syphilitic autopsy material they may remain viable for 26 hours.

Following an injection of penicillin the spirochetes in a chancre gradually disappear after from 12 to 24 hours by a process of fragmentation and cessation of motility.

SEROLOGIC TESTS FOR SYPHILIS

The following tests are used in this country for the detection of syphilis. Some laboratories employ the V D R.L. test routinely. Complement fixation. Standard Wassermann. Eagle Modification Kolmer T P I.

Flocculation. Eagle Hinton Kahn, V D R.L.

Microscopic flocculation. Kline Mazzini V D R.L.

Serologic tests must not be considered to be infallible or used as an excuse from a poor history and a hurried physical examination. Their value is dependent on the reputation of the laboratory and its technicians.

Quantitative serology is important in the following cases: (1) as a guide to therapeutic response (2) as a means of differ

entiation between relapse and reinfection (3) to detect impending clinical relapse and (4) to detect masked syphilis in penicillin-treated gonorrhea etc.

Treatment based on a single positive serologic examination is unfair to the patient in the absence of clinical evidence of syphilis. If the tests disagree in a patient who never has had treatment for syphilis the results should be checked with other laboratories, and a more detailed physical examination should be made.

False-positive reactions occur in diseases where there is an alteration in the globulin fraction of the plasma. Quantitative tests showing a continuous drop in the titer of the serum are strong evidence in favor of a false serologic reaction. However persistence of the positive serology beyond 6 weeks by any test, in the continued absence of any of the following causes should arouse suspicion of syphilis. The following diseases and conditions may produce false-positive readings active tuberculosis, acute lupus erythematosus malaria, diabetes infectious mononucleosis lymphogranuloma venereum leprosy yaws, relapsing fever smallpox vaccination pinta rat bite fever hyperproteinemia, virus pharyngitis and virus bronchitis, periarthritis nodosa, trypanosomiasis and infectious jaundice.

MANAGEMENT A multiple test battery should be made. Serologic discord and low titers are suggestive of nonspecificity. In addition the TPI (Treponema Immobilization Test) should be made in every case in which a false-positive serologic test is suspected. This test is performed by the Venereal Disease Research Laboratory, Chamblee Ga.

The TPI test is used to distinguish a true syphilitic from a false-positive serologic reaction. The test is based on the fact that in the presence of active complement the mobile treponemes obtained from rabbits testicles are immobilized if demonstrable amounts of antibody are present in the patient's blood. It is not a criterion for cure since antibody may persist for many years.

TREATMENT No treatment should be given for at least 3 months of serologic study.

PATHOLOGY

The intrinsic changes in the tissues are similar in the various stages of syphilis the difference is merely one of degree. Immediately after the *T. pallidum* enters the skin or the mucous membranes and penetrates the perivascular lymphatics, a local-

ized vascular dilatation occurs. This is followed immediately by a tissue response consisting of a perivascular infiltration of lymphocytes, plasma cells and connective tissue cells. Many of the spirochetes leave the lymphatic channels to escape into the blood stream momentarily and to lodge in other tissues to set up focal lesions.

In primary syphilis, the chief features are acanthosis of the epidermis, edema of the cutis, followed by fibrosis, and an infiltrate of plasma and lymphocytes about the lymphatics and the capillaries. Small areas of necrosis may result from obliterative changes in the capillaries.

In secondary syphilis, the macular lesions may not show any diagnostic features, but the papular lesions present a perivascular "coat sleeve" infiltrate and a marked endothelial edema of the capillaries (Lever).

In late syphilis of the skin the chief changes are in the dermis which presents a marked infiltrate of plasma and lymphocytic cells, foreign body-type giant cells and caseation necrosis. The epidermal changes consist of pseudoepithelial hyperplasia.

Vascular changes are present in all stages of syphilis eventually leading up to an obliterative endarteritis. This pathologic change produces focal necrosis, the seriousness of which depends upon the tissue affected.

ACQUIRED SYPHILIS

PRIMARY SYPHILIS

After exposure and subsequent inoculation an incubation period occurs during which there is no clinical evidence of the disease. However at this time there is an active dissemination of the infection by way of the lymph and the blood streams. This dissemination is continued throughout the primary and the secondary stages. The incubation period averages about 21 days but may last for 10 days to 3 months. Early syphilis may be masked and delayed by the use of penicillin in cases of mixed infection with gonorrhea. These patients should be under clinical and serologic observation for at least 3 months following discharge.

Chancre. Clinically the primary stage consists of a cutaneous reaction at the site of inoculation resulting in the chancre the primary or initial lesion. This lesion may occur on any accessible portion of the body. Since infection usually is acquired through sexual intercourse about 94 per cent of all chancres

occur on the genitalia. Inoculation is through a microscopic break in the continuity of the skin or the mucous membranes.

The chancre is a hard dull-red painless lesion with a characteristic cartilaginous induration. A slight discharge may be present in the ulcerative types. The draining lymph nodes are enlarged and painless and resemble a chain of beads. In many of the cases a cordlike infiltration of the draining lymph vessels can be felt by palpating the dorsum of the penis. This infiltration reaches up to the symphysis pubis. Multiple chancres may be present if multiple inoculation has taken place.



FIG. 113. Chancre of the penis.

VARIETIES. There are four types of chancre (1) the hard papular (2) the ulcerative or Hunterian which has a granular floor (3) the erosive which is characterized by serous secretion and crusting and (4) the plaque variety which is flat and has a cardboard induration.

The chancre when it is present on the male genitalia, may involve the following: corona prepuce, glans, meatus, shaft, dorsum or root. In the female vulva fourchette labia minora or cervix are common sites. Chancres of the cervix are more common than statistics indicate. Because of their small size and the associated edema and inflammation, they are apt to be overlooked even by experts. In women the primary lesion is usually smaller and often escapes detection especially in pregnancy.

Endo-urethral lesions are often associated with gonorrhea and comprise about 4 per cent of all genital chancres. The presence of induration which is felt upon horizontal and vertical

palpation and an associated inguinal adenopathy should make one suspicious. The diagnosis can be confirmed by endoscopy.

EXTRAGENITAL CHANCRES are usually diagnosed later than genital lesions. From about 6 to 8 per cent of all chancres are extragenital.

Lip chancres are eroded or thickly crusted and characterized by marked local adenopathy. Herpes of the lips lasting over two weeks and associated with unilateral adenopathy should make one suspicious of a chancre.



FIG. 114. Chancre of the prepuce (Dr. A. W. Nelson's case).

Chancres of the tongue which usually occur on the dorsal surface, are eroded, painless and surrounded by an edematous area. Figure 116 illustrates the infrequent sclerotic type.

Chancres of the fingers may resemble a mild paronychia or an angry inflammatory and ulcerative lesion. It is advisable to perform a darkfield examination on any lesion of the fingers which does not heal within 2 weeks under standard antiseptic therapy.

To sum up the clinical characteristics of extragenital chancres are slow development, induration and localized adenopathy. The latter may be more evident clinically than the initial lesion. The secondary eruption may or may not be present at the time of the examination. It is often difficult to get a history of exposure from



FIG. 115 (Left) Seropositive chancre of the lip. The submental lymph nodes were enlarged.

FIG. 116 (Right) Sclerotic chancre of the tongue.

the patient so that the diagnosis must be based on the darkfield examination.

Diagnosis. The diagnosis of any genital lesion is purely a laboratory problem. A darkfield examination is indicated at the time of the first consultation and if negative should be repeated within 24 hours. It is unwise to depend upon the patient's story or the clinical characteristics, for they are not always reliable.

Serologic tests are of little value in differential diagnosis, as they are often negative during the first week following the appearance of the chancre.

A *seropositive chancre* is one in which the serology is already positive. A *seronegative chancre* is one in which the dark field examination is positive but the serology still is negative.

DARKFIELD EXAMINATION. If local treatment has been used, a normal saline-solution dressing should be applied for 24 hours before the darkfield examination is made. This permits the lymph containing the spirochetes to reach the surface. If the lesion has been treated locally or if the patient has received antibiotics, the darkfield examination usually will be negative and should be repeated.

PATHOLOGIC VARIATIONS. *Syphilis d'emblee* is an infection developing at the site of inoculation without the appearance of a chancre. This rare condition may follow pin-prick punctures of the skin, dental operations and blood transfusions.

In *transfusion syphilis* the secondary eruption does not occur until about 4 weeks later. This unfortunate complication can be prevented by insisting upon (1) a serologic test on all donors whether related or unrelated to the patient (2) a physical examination at the time of the transfusion including examination of the genitals for chancre, of the mouth for mucous patches, and of the skin for a cutaneous eruption.

Reinfection is the development of a new primary lesion in a patient who has been "cured" of his original infection. Bernard's criteria for a diagnosis of reinfection are the following: (1) the diagnosis of the first infection must be unquestionable (2) the treatment must have been thorough and adequate (3) the verification of cure must be confirmed subsequently by a reactivation test (4) there must be a sufficient interval between the end of the treatment and the appearance of the second chancre (5) the diagnosis of the second chancre must be confirmed and (6) both infections must have been observed by the same physician or by two competent physicians.

Superinfection is the reinoculation of the patient with the *T pallidum* during the course of the disease. This rare phenomenon of localized immunity usually results in the appearance of a lesion typical of the stage of the original infection.

Chancro redux (chancriform gumma) is the presence of a gumma in late syphilis at the site of the original chancre.

Mixed chancre is a primary lesion complicated by the presence of an infection with Dickey bacilli.

Differential Diagnosis CHANCROIDS are usually painful soft, moist multiple, actively spreading and associated with a painful inflammatory inguinal adenitis. The incubation period varies from 1 to 4 days. The primary lesion is a papule, becoming papular then pustular and finally becoming an ulcer with undermined edges and an irregular border. Smears (methyl green pyronine) and cultures (blood agar) are usually unsatisfactory but the auto-inoculation and the Ito-Reensterna tests are usually reliable. It is not advisable to rule out the possibility of syphilis until after 3 months of observation. No caustics or local medication should be used until repeated darkfield examinations have been made on the lesion for the draining lymph nodes. The Kolmer test should be repeated weekly for 4 weeks and then monthly for 3 months.

HERPES PROGENITALIS may offer confusion. Herpes usually has a short incubation period. There is often a history of repeated occurrences. The disease is associated with burning or itching and the absence of inguinal involvement.

SCABIES. The pruritic papulopustules of scabies practically always are associated with similar lesions on the buttocks the trunk and the upper extremities.

LYMPHOGRANULOMA VENEREUM is characterized by a small evanescent herpetiform primary lesion in its early stage and typical lymph-node involvement. The Frei test is positive.

FURUNCLE is diagnosed by a history of an acute onset, pain and the presence of suppuration after a few days.

"**HAIRCUT**" is a transient superficial penile abrasion often traumatic. It is rhomboidal in outline and responds to soothing local therapy.

Course. Spontaneous involution occurs without treatment after from 3 to 12 weeks usually with permanent scarring. Following treatment with penicillin the lesion usually disappears within 10 days.

Treatment. See Treatment of Early Syphilis, page 343.

SECONDARY SYPHILIS

After the primary lesion appears there is a second incubation period before the appearance of the secondary lesions. This may last for from 2 to 6 weeks or for as long as 6 months. The chancre may or may not persist during this period.

The secondary stage is the stage of active dissemination. Together with the primary lesion it constitutes that infectious phase of the disease called early syphilis. Cutaneous and mucous-membrane lesions occur in about 80 per cent of the cases.

Although the spirochetes invade the blood stream early they do not remain there long because the spirochete is an anaerobic organism. The type of eruption which appears during the secondary stage depends upon the virulence and the number of organisms, the resistance of the host and the "soil," or the type of skin. Those lesions occurring late in the secondary stage are called intermediate lesions. These are usually annular or corymbous. Serologic blood tests are strongly positive in from 99 to 100 per cent of the cases during the secondary stage.

Clinical Types. **MACULAR SYPHILID (ROSEOLA)** This is a generalized symmetrical faint red or pink macular eruption with-

out perceptible infiltration, scaling or itching. It appears most frequently on the abdomen, the flanks, the shoulders, the upper arms and the back.

The macular syphilid must be differentiated from mild erythema multiforme, pityriasis rosea, drug eruptions and typhus fever.



FIG. 117 Early macular syphilis. (Dr. Leon Goldman's case)

MACULOPAPULAR SYPHILID. This is a macular eruption interspersed with papular elements. There is a moderate infiltration present. It may be mistaken for acute psoriasis, pityriasis rosea or seborrheic dermatitis.

Differential Diagnosis. Pityriasis rosea is a superficial condition that occurs along the lines of cleavage of the skin. Even in the papular types there are frequently a few of the typical oval lesions with marginal scaling which are so characteristic of the disease.

PAPULAR SYPHILIDS are coppery-colored infiltrated lesions. They consist of several types, including the following:

Small Papular (Viliary). These are grouped indolent dark-red or brownish papules resembling lichen planus, acne vulgaris or keratosis pilaris. Other signs of syphilis are usually present.

Large Papular (Lenticular) These are large flat round or oval coppery infiltrated lesions.

PSORIASIFORM SYPHILIDS are rare, scaly infiltrated, plaquelike lesions resembling psoriasis. They are apt to occur in psoriatic patients or in those who give a previous history of seborrheic dermatitis. In most of the cases typical maculopapular syphilids are also present.

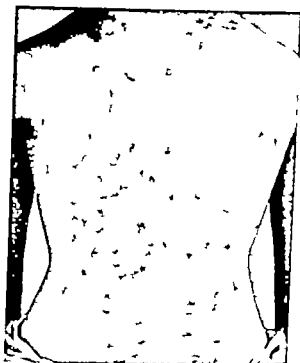


FIG. 118 Maculopapular early syphilis

DISCOID OR EROSIVE, SYPHILIDS (MOIST PAPULES) This is a large papular syphilid which has lost its stratum corneum as a result of moisture and friction. In certain individuals proliferative changes occur with the production of flat condylomata. They are slightly raised, moist mildly inflammatory eroded painless, round or oval and covered with a grayish membrane. This type of lesion is found in the axillary spaces, the gluteal folds and the inguinal region the commissures of the mouth and on the penis.

It is seen commonly in Negroes. Since the raw surface of the papule is exposed, the lesions are very infectious. The discoid syphilid must be differentiated mainly from pemphigus vegetans, but this is easy if the history the character of the lesion and the presence of grave constitutional involvement are taken into consideration.



FIG 119 Rupial (erythematous) syphilis.

PUSTULAR SYPHILIDS are rare and usually occur in Negroes. They have a predilection for the face and the scalp.

The miliary (follicular) type is somewhat pruritic and scaly. There is a tendency toward grouping. The trunk and the scalp usually are affected. It must be differentiated from follicular trichophytids, the follicular seborrheid and lichen scrofulosorum.

The varioliform syphilid resembles variola which is characterized by constitutional symptoms, fever and leukocytosis. The presence or the remains of a primary lesion and a strong positive

serology help to differentiate the two diseases. This type also may be mistaken for a pustular acne or an iodide and bromide eruption.

The erythematous (ruptal) syphilid occurs as a crusted, ulcerative usually multiple lesion on the trunk and the extremities in patients with lowered resistance (See Fig 119)

LATE SECONDARY (INTERMEDIATE) SYPHILIDS

The annular syphilid which is frequent in Negroes, affects the region about the mouth and the nostrils. The lesions may consist of a ringed form a target pattern or only a segment of a circle. The face and the back usually are involved. Annular Eichen planus lupus erythematosus, seborrheic dermatitis and psoriasis



FIG. 120 Annular syphilid in a Negro. This is a late secondary eruption.



FIG 121 (Left) Labial mucous patch (Right) Mucous patches of the tongue

are differentiated from it by the involvement of other areas with typical lesions, by the history the negative serology and the course of the disease. (See Fig. 120)

The corymbose syphilid is a grouped lesion consisting of a central papule surrounded by satellite lesions.

LEUKOMELANODERMIA OF THE NECK (Collar of Venus) is a pigmentedary syphilid consisting of a mottled lacework patch on the side of the neck or the upper chest. The eruption is not infrequent in brunette women. It occurs from about 4 to 6 months after the onset of the primary infection

RECURRENT ROSEOLA is a rare intermediate lesion, more localized in its distribution sometimes annular in configuration and characterized occasionally by a brownish color

PALMAR AND PLANTAR SYPHILIDS are asymmetrical grouped, deep papular lesions with a smooth rough scaly or fissured surface. They must be differentiated from psoriasis and dermatophytosis.

GENERAL CHARACTERISTICS OF SECONDARY ERUPTIONS.

Color—pink to coppery

Arrangement—symmetric.

Grouping—discrete grouped, annular or corymbose.

Itching—absent except in follicular types.

Involution—no scarring except in large papular and pustular types when pigmentation or macular atrophy may follow.

MUCOUS-MEMBRANE LESIONS.

Mucous patches are erosive syphilids which have coalesced to form patches. They may occur on any mucous surface, including lips, tongue buccal mucosa, pharynx, larynx vulva vagina, prepuce and anal region. The lesions are painless, oval or round, mildly inflammatory slightly raised and covered with a grayish membrane. Four types are described the erosive, the papulo-erosive the hypertrophic erosive (condyloma) and the ulcerative. These must be differentiated from the mucous-membrane lesions of erythema multiforme lupus erythematosus, lichen planus, pemphigus and herpes simplex.

Condylomata lata or vegetative hypertrophies, are flat raised, moist, mushroom or cauliflower lesions with eroded surfaces. Since they are very vascular and contain numerous spirochetes, they are very infectious lesions. They usually occur about the anus the inner thighs, the groins or the vulva. The diagnosis is made by the clinical appearance of the growth and darkfield examination of the serum obtained from the base of the lesion after thorough cleansing. Differential diagnosis must be made from hemorrhoids and verruca acuminata.

Laryngitis erosive tonsillitis and pharyngitis are not uncommon. Laryngitis is accompanied by persistent hoarseness. Syphilitic sore throat is characterized by slow onset, absence of acute constitutional symptoms and resistance to local treatment. The patches on the tonsillar pillars show more infiltration and erosion than they do destruction. The draining lymph nodes are relatively large in proportion to the size of the tonsillar lesion. In every case of suspicious sore throat it is advisable to look for a generalized rash and to examine the blood for syphilis. The physician should protect himself against exposure.

SYPHILITIC ALOPECIA is the result of a follicular syphilitic eruption in the scalp and is characterized by numerous small moth-eaten patches of partial alopecia. The condition usually begins at the posterior hairline. The eyebrows also may be affected. As a general rule the condition is temporary with regrowth after 6 months. A diffuse alopecia is also fairly common in early syphilis in both sexes, with general thinning of the scalp hair. This is the result of a toxic constitutional condition.

CONSTITUTIONAL SYMPTOMS may occur in some cases as a toxic reaction. These include fever sore throat, headache malaise loss of weight, joint pains and general adenopathy



FIG. 122 Syphilitic alopecia. Note moth-eaten appearance.

CENTRAL NERVOUS SYSTEM INVOLVEMENT may develop early in the disease. If the spinal fluid is examined after the second course of treatment in early syphilis, many of the precocious cases will be discovered at that time.

Symptoms in premature neurosyphilis include persistent headache stiff neck acute neurasthenia mental confusion convulsions, weakness, numbness or paralysis of the extremities, tinnitus impairment of hearing vertigo faulty vision or diplopia.

TREATMENT OF EARLY SYPHILIS

The purpose of treatment in early syphilis is to complete the prescribed treatments, to "cure" and to eradicate the disease

completely and to control the infectiousness of the patient. With adequate penicillin treatment 100 per cent "cures" can be obtained in seronegative primary syphilis, 90 per cent "cures," in seropositive primary syphilis and 85 per cent "cures," in early syphilis. Adequate treatment is also important in the prevention of mucocutaneous (infectious) ocular and neurorecurrences (relapses) which may occur from two years or longer following the cessation of treatment.

Instructions to Patients. Patients should be advised to avoid excesses in all things. Kissing and sexual intercourse should not be permitted until six months have elapsed after beginning treatment when the blood and the secretions are usually no longer infectious. As a public health measure all contacts as well as the marital or the sex partner should be examined for evidence of the disease.

Premarital Examinations. A negative serology does not rule out early syphilis in the seronegative stage, so that a general inspection is also necessary. If one of the parties has clinical or serologic evidence a total of 5 million units of penicillin should be given. If the bride has had syphilis and becomes pregnant, she must take penicillin therapy during the first trimester to protect the fetus. Syphilis is rarely infectious when over 5 years old.

Local Treatment. Excision of the chancre is a useless procedure since it is well known that by the time of its appearance the infection already is disseminated throughout the body. All that is necessary in the management of infectious lesions is the judicious use of soap and water. Mixed chancroidal lesions should be treated with applications of hot boric acid or permanganate (1:5,000) solutions.

Acute Iritis which is best treated by the ophthalmologist, can be controlled with a 1 per cent solution of atropine to keep the pupil dilated and thus to prevent adhesions.

Prognosis. Relapses occur in 10 per cent of the cases, and the blood has been serologically fast in 5 per cent of the cases.

PENICILLIN THERAPY. Procaine penicillin in oil with aluminum monostearate is the preparation of choice at present. "One-shot" injections of penicillin are not considered adequate therapy. The average dose used in primary syphilis is 3,600,000 units; in secondary syphilis 4,000,000 units. Cases should be followed up monthly for one year after penicillin therapy with a spinal fluid examination in 18 months.

LATENT SYPHILIS

The latent period, or the stage of temporary or apparent inactivity usually sets in about 5 years after the secondary stage. It may last from a few months to a lifetime, with an average of about 7 years. During this period Nature's defense mechanism is established with the host and the parasite living in harmony with a minimum of pathologic activity. Clinical latency signifies the absence of all clinical recognizable syphilitic lesions; pathologic latency denotes the absence of pathologic activity although spirochetes are present; and serologic latency means a negative blood and spinal fluid in the presence of spirochetal foci.

Etiology. About 50 per cent of all latent syphilitics give no history of infection. In this group two thirds are women and one third are men. A large percentage of these probably had silent, or symptomless, primary infections. About one third of all syphilitics seen in clinic and private practice belong to the latent group. Laboratory studies have shown that about 15 per cent of latent syphilitics have asymptomatic neurosyphilis.

Diagnosis of true latency is based on a positive serologic test in the absence of all positive physical findings, although many latent syphilitics suffer from unrecognized or subclinical cardiovascular syphilis. However there is a tendency for the serologic reactions to fluctuate over a period of years. The diagnosis of latency also is made in those seronegative cases where there is a history of inadequately treated syphilis, or in women, a history of the birth of a syphilitic child. Every case of latent syphilis should have a T.P.I. test to rule out false positives.

Prognosis. An untreated syphilitic has one chance in three of escaping the serious manifestations of late syphilis. About 15 per cent ultimately will develop cardiovascular syphilis, 6 per cent will develop neurosyphilis, from $\frac{1}{2}$ to 1 per cent will have other forms of visceral syphilis, and about 15 per cent will develop late syphilis of the skin and the mucous membranes.

Treatment. The aim of treatment in latency is (1) To prevent infection to others. The blood serum and the lymph nodes have been shown to harbor spirochetes. (2) To prevent infectious relapse. Clinical arrest, not clinical "cure" is the chief aim of treatment. Serologic relapse is of little importance if treatment has been adequate.

THE SCHEME OF TREATMENT includes a minimum of 3.6 million units or more of penicillin, depending upon the response, the severity and the extent of the case.

LATE SYPHILIS

After a third incubation period, which varies from $1\frac{1}{2}$ years to 40 years, the tertiary or gummatous, stage may become clinically manifest. This is the stage of late, localized or restricted activity. A walling-off of the spirochetes produces a characteristic tissue reaction called a gumma. Any organ in the body may become involved, depending on such factors as previous disease, trauma, alcohol, heavy work and race. The gumma is believed to be an allergic reaction to the presence of the spirochetes, the majority of which probably are killed instantly by the immunologic response of the tissues. The organisms are rarely present in the



FIG. 123 Late ulceronodular syphilis. Note the grouping of the lesions (Dr. C. O. Kling's case)



FIG. 124 (Left) Late nodular syphilis of the face. (Right) Late ulceronodular syphilis of the face.

lesions. They cannot be demonstrated by the darkfield and can be shown only seldom in stained sections.

Tertiary Lesions of the Skin. Gummas are characterized by slow growth. They may occur on any part of the cutaneous surface. They are usually single, often are grouped and are asymmetrical and painless. Induration and low-grade inflammation are always present. The nodular and the squamous types have polycyclic punched-out edges. Central or unilateral healing with thin atrophic wrinkled scars surrounded by a hyperpigmented border is characteristic. Serologic tests are negative in less than 10 per cent of the cases. Frequently an adequate biopsy is necessary to establish a diagnosis.

Differential Diagnosis. Ulceronodular lesions on the scalp must be differentiated from carcinoma, lupus erythematosus and lupus vulgaris. Nodular lesions on the face may be confused with epithelioma, sarcoid or lymphoblastomas. Gummas on the extremities may resemble sarcoma, leprosy or sarcoid. Ulceronodular gummas on the hands and the forearms may be mistaken for

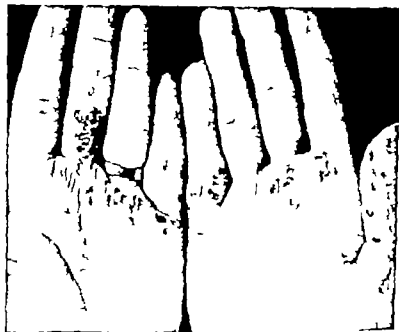


FIG. 125 Verrucous late palmar syphilis. Often confused with ordinary warts. (Dr W. M. Sam's case)

tularemia, sporotrichosis carcinoma or blastomycosis. Squamous or psoriasiform gummas on the trunk must be differentiated from psoriasis, seborrheic dermatitis and superficial epitheliomatosis.

MUCOUS-MEMBRANE LESIONS. The tongue is not frequently involved in late syphilis. *Smooth atrophy of the tongue* consists of smooth atrophic red areas devoid of papillae. This lesion may develop gradually into a chronic interstitial glossitis, or cobblestone tongue. Ulcers and gummas also may be present.

If leukoplakia involves the tongue, the penis or the mucous surfaces of the labia in a syphilitic patient, there is a greater possibility of malignant degeneration than in a nonsyphilitic patient. If the border of the leukoplakic patch is raised, inflamed, eroded or fissured a section should be made at once and examined microscopically for malignancy. Antisyphilitic treatment has no effect upon fully developed leukoplakia, which must be treated by removing all possible sources of irritation.

GUMMAS. The lips, the nasopharynx the larynx the tonsils or the palate may be the site of ulcerative gummas. Perforation of the palate results in regurgitation of food and gives a nasal quality to the voice.

Differential Diagnosis. Tuberculous ulcers are slower in development they are painful shallow and associated with open cases of visceral tuberculosis. Carcinoma is more infiltrated the draining lymph nodes may be enlarged and there is no tendency toward spontaneous healing. In general syphilis should be suspected before the age of 40 while carcinoma should be looked for after the age of 40. A biopsy should be taken in doubtful cases.



FIG. 126 (Left) Multiple ulceronodular gummas of the leg.
FIG. 127 (Right) Perforated palate (a result of healed gumma)

TREATMENT OF LATE CUTANEOUS SYPHILIS

General. Treatment should not be undertaken without a complete physical examination to determine the presence of visceral syphilis and the state of the cardio-vascular system. Intensive sterilization technics are not used for two reasons (1) the possible danger of a serious Herxheimer reaction and (2) the fact that symptomatic cure not serologic, is the primary aim. Because of the possibility of coexisting visceral pathology the treatment must be individualized. Routine therapy has no place in late syphilis.

Iodides are always necessary in this stage to absorb the granulation tissue. Starting with 5 drops of a saturated solution of potassium iodide the dosage is stepped up daily until the patient is taking 30 drops three times a day after meals. If he is intolerant to the oral administration sodium iodide should be injected intravenously (from 15½ to 120 grains twice weekly).

To avoid a Herxheimer focal reaction it is important to administer iodides for at least 2 weeks *before* administering antibiotic therapy. The patient should be observed for jaundice, eye cardiac and neurologic signs.

Drug Fastness. Those cases of late syphilis which do not respond to penicillin should be given some form of fever therapy to break down the resistance of the spirochetal foci in the diseased tissues.

Local therapy is not necessary although a mild antiseptic lotion or ointment is sometimes useful psychologically.

Penicillin Therapy. Late syphilis requires 10 000 000 units of penicillin. The initial dose should be 30,000 units to avoid a Herxheimer reaction. Iodides or fever therapy may be given *col* laterally depending on the type of case and the organ involved.

CONGENITAL SYPHILIS

Probably infection of the fetus does not occur until the fourth month of pregnancy. Congenital infection may result in three possibilities. (1) a miscarriage (2) a stillbirth or (3) a living child who may develop early or late manifestations of the disease. Third-generation syphilis is rare.

Fetal Syphilis. If the disease results in the death of the fetus, there are marked placental changes. The placenta is pale soft, friable and spotted with yellow patches. Postmortem findings in the fetus consist of an enlarged liver and spleen, chondro-

epiphysealitis and interstitial pneumonia. The fetus has a characteristic "boiled skin," which is shriveled and macerated and contains hemorrhagic bullae. The cord and the maternal serology are usually positive.

CUTANEOUS MANIFESTATIONS OF CONGENITAL SYPHILIS Contrary to general opinion the bullous syphilid is a rare lesion in early syphilis. It is confused frequently with impetigo contagiosa bullosa and epidermolysis bullosa.

The *maculopapular syphilid* which is the most common type of eruption, has all the characteristics of the acquired variety



FIG. 128 Bullous syphilids (early prenatal stage)

In most of the cases the coppery color and the distribution about the mouth, the anogenital region and the palms and the soles is a diagnostic feature. The maculopapular types are confused occasionally with the nevroid condition *urticaria pigmentosa*. This disorder consists of persistent pigmented macules or nodules which urticate upon rubbing.

Mucous membrane lesions consist of condylomata and mucous patches about the mouth and the anogenital region.

Fissures which ultimately leave radiating scars (rhagades) may develop about the mouth and the lips.

Sneezes due to a rhinitis appear early. In some cases the discharge may be hemorrhagic.

Dactylitis is fairly common in the first year or two of hereditary syphilis. It is characterized by a fusiform thickening of a proximal phalanx, the result of periostitis.

Cracked cry is distinctive.

Osteochondritis and *chondro-epiphysitis* may simulate paralyzes (Parrot's pseudoparalysis).

Craniotabes consists of softening of the cranial bones from thinning.

LATE CONGENITAL SYPHILIS usually makes its appearance at 12 years of age with activity ceasing at the age of 30 or soon after. The diagnosis often is made during the course of a physical examination for some other condition. According to Stokes, the six most common signs are (1) eye lesions, (2) positive serology (3) bosses, (4) sabre shins, (5) characteristic teeth and (6) saddle nose. In his series of 202 cases, 26 per cent had neurosyphilis and 20 per cent had abnormal spinal fluids.

Cardiovascular changes are relatively infrequent.

Facies The characteristic change in the appearance of the face is often of diagnostic value and should awaken suspicion. The gross changes include prominent frontal and parietal bosses (proliferative periostitis) saddle-bridge nose (result of bone destruction) and narrowing of the face to the chin (bee face or pear face). Minor characteristics include prominent supraorbital ridges, increased distance between the eyes and a deepening of the eye sockets.

Eye Lesions Interstitial keratitis is the most common eye lesion followed by choroiditis and iritis. Keratitis always leaves some cloudy scarring. It frequently recurs from insufficient treatment and must be differentiated from tuberculosis. Eye conditions should be treated and examined frequently by the ophthalmologist.

Eighth nerve deafness occurs in about 10 per cent of the cases and usually is preceded by tinnitus and vertigo. The condition usually develops at puberty. Together with interstitial keratitis and dental deformities it constitutes the so-called "Hutchinson's triad."

The Blood Serology in Congenital Syphilis Because of the possibility of false-positive or negative serologic tests at birth, the blood should not be tested before the tenth day. Positive serologic reactions are at their peak in the second and the third months of the disease when the clinical manifestations are promi-

ment. After the second year less dependence should be placed upon the serologic tests.

Dental signs are found in about one third of the cases. The first set of teeth have a low vitality. Only the teeth of the second dentition show the characteristic changes, which can be detected occasionally by roentgenograms taken before they erupt. The upper central incisors, which bear the brunt of the aplasia, are affected by two types of changes. (1) pegging with central notching (Hutchinsonian teeth) and (2) narrowed cutting edge with lateral beveling (screw-driver type). Mulberry teeth involve



FIG. 129 Severe congenital syphilis with involvement of the frontal and the nasal bones, the palate and the eyes. Fatal case.

the 6-year molars and bear dwarfed defective cusps surrounded by a shoulder of enamel which bulges out around the crown. Malocclusion and faulty spacing of the incisor teeth are common suggestive findings but are not pathognomonic signs.

Bones Saber tibiae which are characterized by a combination of anterior bowing and thickening are a result of osteitis and periostitis in the early stages of the disease. An enlargement of

the inner third of the clavicle is considered to be of diagnostic importance. In some cases there is epiphyseal enlargement of the joints, giving rise to thick knees and wrists. Dactylitis is uncommon in late prenatal syphilis.

Another frequent sign of late congenital syphilis is symmetrical hydrarthrosis (Clutton's joints) which is characterized by serous effusion and enlargement of the knee or the elbow joints.

A high narrow palatine arch is not diagnostic of congenital syphilis but is merely suggestive. Perforated nasal septum and perforation of the hard palate are not uncommon. The former is

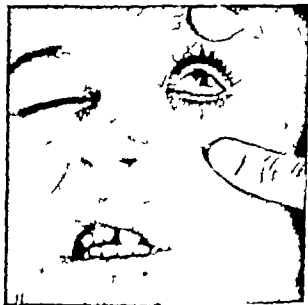


FIG. 130 Late congenital syphilis. Hutchinson's teeth and interstitial keratitis.

the result of a gummatous osteitis of the nasal septum and results in a saddle bridge.

Scaphoid scapula is considered to be a stigma of constitutional inferiority and is found in various degenerative conditions, including syphilis.

Rhagodes are radiating scars about the mouth and the lips, resulting from healed infiltrated and erosive syphilids.

Visceral disease is uncommon. The liver and the spleen are palpable in about 20 per cent of the cases. Syphilitic children are usually subject to colds, pneumonia and various minor ailments.

Juvenile tabes and paresis (positive spinal fluids in 15 per cent of the late cases) often are detected in their early stages by routine examination of the spinal fluid. The juvenile types are not so severe as the adult types of involvement. Nervous irritability, backwardness, precocity, conduct disorders and retarded growth are met with occasionally in some cases.

TREATMENT OF CONGENITAL SYPHILIS

Infants born of treated syphilitic mothers and apparently normal should have weekly serologic tests for 4 weeks. A final test should be made at the age of 6 months. If the infant's serology is positive and if the titer persists at high level or increases with subsequent tests after 4 weeks of observation treatment should be started at once.

Early Congenital Syphilis (under 2 years of age) The aim of treatment is similar to that of early adult syphilis, i.e. radical "cure."

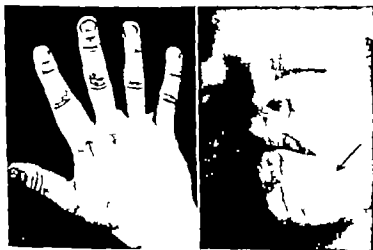


FIG. 131 (Left) Dactylitis resulting in diffuse fusiform enlargement of the digits

FIG. 132 (Right) Rhagades in congenital syphilis, resulting from healed fissures about the mouth

Penicillin Therapy The total dosage for the first course is 200 000 units of aqueous penicillin per kilogram of body weight, in equally divided doses given every 3 hours over a period of 10 days. The first 5 injections should be 30 000 units each in order to avoid a Herxheimer reaction. Seronegativity usually occurs in from 4 to 12 months in about 85 per cent of the cases. Clinical or serologic relapse within 6 months after therapy calls for another course of penicillin (of double the original dose).*

Late (tardive) congenital syphilis (over 2 years of age) is treated with 600 000 units of procaine penicillin, given every other day for a total of 10 injections.

Relapses should be treated at double the original dose.

Supportive treatment is necessary also to combat anemia and low resistance.

Serologic relapse occurs in about 15 per cent of the cases.

SYPHILIS IN PREGNANCY

Congenital syphilis is a preventable disease and can be eradicated if physicians will perform a routine serologic test on every pregnant mother and if they will administer adequate treatment to all syphilitic mothers during early pregnancy. Since the fetus becomes infected at the fourth month or soon thereafter treatment should be instituted as early as possible. Syphilis is much milder during pregnancy so far as clinical evidence is concerned. The pregnant state protects the mother against severe late manifestations and central-nervous-system invasion.

The Kahn and V.D.R.L. tests in pregnancy are completely reliable. In the absence of a positive history and clinical findings, penicillin therapy should be given to protect the fetus. After delivery a battery of serologic tests including the T.P.I. test, provided that no oral or injected antibiotics have been given within one month, should be made on the mother's blood to prove or disprove the diagnosis.

TREATMENT

Penicillin is the best therapy for syphilis in the pregnant woman because of ease of administration, freedom from reactions, and prevention of prenatal syphilis. The minimal dose is 4,800 000 units, administered during the first trimester if possible. A

*For a more complete review of the subject, see Moore J. E. Penicillin in Syphilis, ed. 2 Springfield, Ill. Thomas.

second course is necessary if the serology does not decline after 3 months or if there is clinical or serologic relapse. Early untreated syphilis in the last trimester requires 10,800,000 units.

The infant should be observed clinically and serologically for 6 months after birth.

SERORESISTANT SYPHILIS

Seroresistant syphilis is that type of infection the serology of which resists reversal to negative in spite of adequate treatment. The incidence is greatest in late congenital late hepatic syphilis and paresis. Early seroresistant syphilis (infection less than 2 years old) is that type which remains serologically positive after 6 months of adequate treatment. When this occurs, prolonged therapy is indicated. Late seroresistant syphilis (infection over 2 years old) is that type in which serologic tests are still positive after one year of continuous therapy.

Etiology Seroresistance may be due to the sensitivity of modern serologic technics. Persistent spirochetal foci, extensive cerebrospinal cardiovascular and osseous involvement and inadequate treatment of early syphilis are also possible causes.

Treatment consists of 6,000,000 units of procaine penicillin.

TREATMENT RESISTANT SYPHILIS

Treatment-resistant syphilis is an infrequent type of syphilis which is characterized by lack of response of the clinical lesion to adequate treatment. It may occur at any stage or involve any organ in the body. In many of the cases the serology is negative. Not infrequently spirochetes persist in the lesions in spite of adequate therapy.

Treatment of first choice is penicillin with some form of fever therapy. A total dosage of at least 10,800,000 units is necessary. Liver and iron medication to improve the general health will be found beneficial.

ANTIBIOTICS

Penicillin Therapy Procaine penicillin G is the drug of choice in the treatment of syphilis because of the ease of administration, the relative freedom of reactions and the good clinical response. Inquiry should first be made regarding a history of penicillin allergy so that adequate precautions can be made or another antibiotic considered. It is not possible at this time to state whether

permanent cures are possible. The optimum dosage appears to be 3,600,000 units in primary syphilis, 4,800,000 units in secondary syphilis, latent, late and serologic fast cases and 10,800,000 units in central nervous system syphilis.

The timing of the injections is important in order to obtain an adequate blood level throughout the period of therapy and to interfere with the 30-hour division-time of the spirochetes.

Patients with early syphilis treated with penicillin often exhibit the following manifestations of the Herxheimer reaction: fever, chills, flareup of lesions, headache, nausea, arthralgia and malaise.

The initial doses should be small to avoid a Herxheimer reaction.

The following methods are suggested:

1. Daily injections of 600,000 units of procaine penicillin G in oil with 2% aluminum monostearate for 6 days or injections of 600,000 units every other day. The first dose should be 1,200,000 units in case the patient does not return for further therapy.

2. Administration of 600,000 units three times weekly for 2 weeks.

Allergic Reactions. These occur in about 5 per cent of the cases. The local reaction is usually a painful edematous nodule at the site of injection. The systemic reactions, which may be caused by a protein conjugate in the tissues, consist of a generalized urticaria, macular eruption, vesicular "id" eruptions on hands, feet or groin, purpura, erythema multiforme or in rare cases glossitis or exfoliative dermatitis.

When penicillin cannot be used because of severe reactions, the following antibiotics may be used:

Aureomycin Therapy. Aureomycin appears to be active against syphilitic infections but it is too early to evaluate this antibiotic in the treatment of syphilis. Frequent gastro-intestinal upsets often interfere with the regularity of treatment. An initial priming dose of 2 Gm. is given and repeated 4 hours later. Then 1 Gm. is given every 4 hours around the clock until a total of 70 Gm. is reached.

Terramycin Therapy. Clinical healing of lesions occurs when the antibiotic is given orally in doses of 60 mg. per kilogram of body weight per day.*

*See Ingand, S. and Alexander, E. R. Treatment of early syphilis with Terramycin, *Am J Syph* 37:247, 1953.

THE PUBLIC HEALTH ASPECTS OF SYPHILIS

A gradual downward trend until recently in the incidence of infectious syphilis has been noted since 1936 as a result of mass education, more available clinical facilities, better trained physicians and the introduction of penicillin. Free treatment centers and the free distribution of antibiotics to physicians also have increased the number of cases reported. About 150,000 new cases of infectious syphilis occur in the United States each year but probably half of them remain undetected and unreported. These patients are the sources of new infections which challenge the ultimate complete control of the disease. In 1953 the number of reported cases of syphilis throughout continental United States (all stages) was 156,099.

While the total number of patients with syphilis in the United States is unknown, the prevalence of the disease has been determined by mass serologic surveys of racial, community, economic, occupational and institutional patient groups. The incidence is higher in Negroes of comparable age, sex and economic status than in whites. Among the whites, the incidence is higher in females. The Negro morbidity rate is the same in both sexes but females acquire the disease earlier. In 1953 3.4 per 100,000 deaths in U. S. A. were due directly to syphilis with a mortality rate three times higher in Negroes. These deaths are caused by aortic aneurysm, paresis, tabes dorsalis and cerebral hemorrhage or thrombosis.

Premarital examination laws vary in the different states but they have not been effective in practice. The intent of the laws is to delay marriage if one of the parties has an infectious or a potentially infectious type of syphilis, until adequate treatment has been instituted. The degree of infectiousness must be based on the history, a complete physical examination, darkfield and serologic studies. It must be emphasized that a negative serologic examination occurs in seronegative primary syphilis and that a false-positive blood may be the result of any of several systemic conditions.

In regard to infectiousness, all early types are infectious, early latent syphilis is potentially infectious, latent syphilis of 5 or more years duration is not infectious (except in utero) and late syphilis is noninfectious. All infectious cases should be treated as soon as the diagnosis is verified.

Trained social service workers are a necessary adjunct in treatment centers to trace contacts, to reduce clinic absenteeism, to help to solve social and economic problems and to educate the patient to co-operate with the medical authorities. Public health officials can help to reduce the incidence of syphilis by taking their departments out of politics by carrying on an unrelenting campaign against syphilis by co-operating with police departments to wipe out prostitution and by aiding the private physician by providing dependable serologic and diagnostic facilities and free penicillin for destitute patients.

NURSING ASPECTS

Syphilis is not so common as formerly in dermatologic practice but patients in all stages are being treated in special syphilis clinics treatment centers and hospital clinics. The percentage of syphilis cases in private practice is not over 2 per cent, with a gradual decline during the past 5 years.

The Examination. Primary and early and early latent cases of syphilis are infectious. If the nurse pricks her skin accidentally with a needle used in such a case the incident must be reported to the physician at once. Equipment for the examination includes reflex hammers rubber gloves free of pinpricks, tongue blades a flashlight blood test tubes, sharp intravenous needles and a few medium-sized hypodermic needles for use in fat individuals with small veins, alcohol sponges tincture of iodine, tourniquets and smelling salts. Special equipment for darkfield examinations of primary lesions may be necessary although these usually are performed by specially trained technicians. The examination room should be well lighted preferably by natural light and should not be too chilly. Examination of the spinal fluid requires the following setup sterile towels and sheets, tincture of green soap tincture of iodine 4 per cent povidone solution alcohol sponges, hemostats, sterile rubber gloves, spinal-puncture needles sterile dressings adhesive plaster and sterile collection tubes properly labeled.

Treatment Table. Before administering penicillin, the nurse should question the patient regarding any reactions from previous use of the drug. Rubber gloves protect the nurse against possible penicillin contact dermatitis and give her psychological, as well as actual protection against infection. An adequate supply of penicillin sharp intramuscular needles, alcohol sponges, and epinephrine should be available.

Benign Tumors of the Skin

CYSTS

SEBACEOUS CYSTS

PERIARTICULAR CYSTS

TRAUMATIC EPITHELIAL

CYSTS

MILIUM

MULTIPLE BENIGN CYSTIC

EPITHELIOMA

HYPERTROPHIC GROWTHS

CLAVUS

CALLUSES

KELOIDS

FIBROMATA

LIPOMATA

LEIOMYOMATA

NEUROMATA

GLOMUS TUMORS

MISCELLANEOUS

PARAFFINOMA

GRANULOMA PYOGENICUM

HISTIOCYTOMA

MARKING ASPECTS

BENIGN tumors are overgrowths of tissue normally present in the region from which they spring. Hamartomas are nevold growths which contain embryonal cells or structures and include a wide variety of congenital tumors. It is important to note that some benign growths occasionally undergo malignant changes under certain conditions. Among these may be mentioned cutaneous horns multiple cystic epitheliomas, certain types of nevi sebaceous cysts and paraffinomas. When benign growths are removed a routine biopsy always is advisable to verify the clinical diagnosis.

Pathologically benign tumors are classified according to their origin (1) of the epidermis corns, verrucae, molluscum contagiosum (2) of fibrous tissue keloid, fibroma histiocytoma (3) of fatty tissue lipoma (4) of muscle tissue leiomyoma, myoblastoma (5) of vascular tissue angioma senile angioma granuloma pyogenicum glomus tumor lymphangioma (6) of appendages sebaceous cysts, senile sebaceous adenoma adenoma sebaceum (7) of osseous tissue osteoma cutis (8) of cartilage chondroma (9) of nerves neuroma (10) mixed tumors dermoids.

Most patients consult the physician for removal of benign tumors for the following reasons: cancerphobia, impending marriage, fancied increase in the size or in the morphology of the lesion, recent irritation or improved financial status.*

CYSTS

SEBACEOUS CYSTS

Sebaceous cysts (wens) occur frequently on the scalp, the lobe and the posterior surface of the ear, the face and the back.



FIG. 133 Sebaceous cyst showing central dimple from obstructed sebaceous duct.

As a rule they are single but multiple lesions are not uncommon. The growths are painless unless secondary inflammation from irritation is present. They grow slowly and, after reaching a certain size, remain stationary.

The cysts are round or oval in shape. They have a pillowy feel and a central dimple which represents the opening of the sebaceous gland through which a foul-smelling caseous matter

*For a complete review of the subject and an extensive bibliography see Elder J. J. and Elder W. D. Tumors of the skin, ed. 2 Philadelphia, Lea & Febiger 1951.

may be expressed by local pressure. Usually they appear after puberty.

Pathology The lesion is a retention cyst with an accumulation of sebaceous material and epithelial debris. There is a secondary hyperplasia of the walls. The external capsule consists of compressed connective tissue. About 6 per cent of sebaceous cysts may undergo malignant degeneration (Caylor).

Differential diagnosis. Dermoid cysts contain remnants of hair follicles or sebaceous and sweat glands that are embedded in the skin along lines of fusion or in embryonic clefts.

Treatment consists of careful dissection of the cyst with removal of the entire capsule. If part of the capsule is left behind there is a possibility of recurrence.

PERIARTICULAR CYSTS

These are round opalescent growths containing a straw-colored fluid. They are split-pea sized and usually occur over the articulation of the digits. Probably they arise from the joint capsule. In most of the cases there is a history of arthritis.

Treatment consist of the application of pressure dressings for several weeks and if this is unsuccessful thorough excision.

TRAUMATIC EPITHELIAL CYSTS

These usually occur following injury and are due to the implantation of parts of the epithelium in the deeper structures.

Therapy consists of excision.

MILIUM

Milia (whiteheads) are small white pinhead-sized globular lesions which are found frequently on the face the cheeks or about the eyes. Occasionally they are found on the shaft of the penis. Milia may occur at any age independently or in association with acne seborrhea senile skin epidermolysis bullosa and scars. They are encysted inclusions of horny cells in the epidermis.

Differential diagnosis must be made from senile sebaceous adenoma.

Treatment consists of shelling out the contents of each lesion with the sharp point of a sterile knife.

MULTIPLE BENIGN CYSTIC EPITHELIOMA

This rare nevroid condition (tricho-epithelioma) appears as clusters of firm shiny pearly translucent pink or straw-colored,

pinhead-sized tumors. They are solid appear to be embedded in the skin and rarely ulcerate. They usually appear on the forehead, above the eyebrows, on the nasolabial folds, the cheeks, the chin and the back.

Etiology The disease which is usually congenital or familial affects females, especially those from 30 to 40 years of age. The lesions spring from epidermal cell rests or embryonic hair follicles or sebaceous glands.

Pathology Fingerlike elongated projections of cellular masses with central-cyst formation and rudimentary hair follicles are present. These strands are composed of embryonal cells and appear to have much in common with the cells of the external sheaths of the hair follicles. The central areas undergo colloidal degeneration.

Course. The lesions never disappear spontaneously. In most of the cases they increase slowly in number until growth becomes stationary. Rarely they may become ulcerative or undergo malignant degeneration with the production of basal-cell carcinoma.

Treatment consists of curettement or fulguration of the individual lesions or dermabrasion of the entire face in extensive cases.

CONGENITAL CYSTS

These rare lesions include dermoid, auricular, odontogenic, and thyroglossal cysts.

Treatment is surgical.

HYPERTROPHIC GROWTHS

CLAVUS

Clavus (corns) are circumscribed bony conical hyperkeratoses consisting of concentric lamellae around a central core and occurring over bony prominences. The wide base of the corn projects from the surface of the skin with the core pressing inward against the adjacent structures.

Etiology They result from intermittent pressure by tight footwear or improperly fitting shoes. Soft corns may be associated with exostoses. They occur between the toes and usually are macerated from friction and moisture. They are often the sites of latent fungus infections.

Treatment. Extensive cases require orthopedic consultation. In the ordinary type frequent paring of the lesion or treat-
ment

It with a keratolytic controls its growth but care must be used in patients with peripheral vascular disease to avoid infection. Surgical removal is advisable in all recurrent and painful types. Corns tend to involute when the source of pressure is removed. A favorite prescription is the following:

Salicylic acid	
Lactic acid 88	30
Colloidal, flexible	30.0

3. Paint corns once a day

CALLUSES

These are circumscribed or diffuse hyperkeratotic plaques with wavy uninterrupted lines. They are due to friction or come as a result of occupation. They differ from corns in the absence of a central core. In common with soft corns, they may house pathogenic fungi.

Differential Diagnosis. When they occur on the soles of the feet they may be mistaken for plantar warts which are more painful and exhibit tiny bleeding points when the hyperkeratotic surface is removed. Arsenical keratoses may also be mistaken for calluses.

Treatment consists of removal of the cause, paring the lesion and the use of a 40 per cent salicylic-acid plaster.

KELOIDS

Keloids are irregular fibrous growths arising from a previous injury or scar. Predisposing causes may be racial (dark races), familial, individual or regional (sternal area). Keloids are white, pink or red in color with a smooth shiny surface. The growth usually is raised above the surface. Clawlike projections radiate from the edges of the lesion. There is a tendency for growth to occur beyond the limits of the original injury. Usually there are no symptoms, although occasionally there may be severe itching or pain which may result from a compression of the sensory nerve endings.* When the lesions have grown to a certain size they become stationary.

Etiology. The lesions may follow burns, scalds, slow healing incised wounds, electrosurgery, vaccination, insect bites, comedones, slight or unnoticed injuries. There appears to be an individ-

*The subject is discussed fully by Traister, H. M. and Bader, T. B.: Keloids and hypertrophic scars, *Arch. Surgery* 57: 129, 1948.



FIG. 134 Linear keloid following incised wound (From Dr. E. W. Spradig, Homer Phillips Hospital)

ual predisposition. Theories as to causation include (1) foreign-body reaction, (2) hypersensitivity of vascular endothelium, (3) hormonal influence, (4) biochemical and biophysical effects, (5) fibroplastic diathesis, (6) chemotropic effect of serum on fibroblasts.

Differential diagnosis is chiefly from hypertrophic scars, xanthoma, and sarcoid in Negroes.

Pathology. Sections reveal a fibrous-tissue hyperplasia, more cellular and vascular than normal. The process apparently starts around the blood vessels. The fibroblasts are arranged in parallel

strands. As a result of pressure atrophy the glands and the hair follicles disappear from the picture.

Treatment. Good results occur only if treatment is applied early. Hyaluronidase (150 to 200 units in 1 cc. 2 per cent procaine) injections into lesions under 1 cm. preceding each x-ray therapy may cause involution if the keloid is under 1 year old.

In older and larger lesions, the lesions must be excised and the injections made under the resulting scar weekly for 3 months. The follow-up with x-ray therapy depends on the clinical result.

In no case can the result be predetermined until several months have elapsed after treatment.

FIBROMATA

Fibromata are soft or hard tumors of the skin or the mucous membranes. They may be sessile or pedunculated, and consist of a hyperplasia of the connective tissue fibers of the skin. The following types are included (a) soft, (b) hard, (c) diffuse (large pendulous growths) and (d) neurofibromata (see p 590).

Simple fibromata do not undergo malignant degeneration.

Pathology The tumor is localized to the cutum and consists of closely packed fibroblasts and fibers of connective tissue.

Treatment is surgical. In all cases the patient should have a complete examination to rule out neurofibromatosis.

LIPOMATA

Lipomata are single or multiple tumors consisting of adipose tissue. They may grow to any size and may occur anywhere, although the trunk, the back of the neck and the forearms are favored sites. The lesions are soft, rounded, subcutaneous, lobulated growths and are freely movable.

Pathology The lesions consist of groups of fat cells held together by strands of connective tissue. It is usual for growth to cease after the lesions reach a definite size. Secondary malignant changes are unknown.

Diagnosis is made by the orange-peel sign. Compression of the tumor between the thumb and the forefinger causes the overlying skin to become irregularly dimpled by the downward traction of the subcutaneous vertical trabeculae.

Treatment consists of surgical excision when indicated for cosmetic reasons or discomfort because of location. If the tumor is removed completely, no recurrence results.

Course. Their growth at first is rapid but later after reaching a certain size they become stationary.

Treatment consists of excision, fulguration or cautery destruction. The last is preferable.

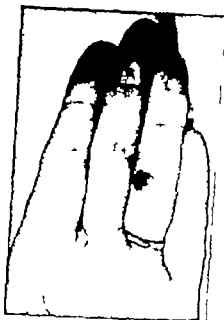


FIG. 135 Granuloma pyogenicum (Dr McCuskey's case)

HISTIOCYTOMA

These are usually solitary benign, hard, fibrous connective-tissue tumors, slow growing intracutaneous or subcutaneous pinkish or tan in color and painless. The lower extremities are the usual site.

Etiology Slight trauma (e.g. insect bites) may be a factor in some cases. They are usually seen in the 30-year-old to 40-year-old age group.

Differential Diagnosis. In fibrosarcoma there is a history of increased growth. The microscopic picture reveals numerous immature cells and mitoses.

Pathology Hemosiderin is usually present, with varying amounts of fibrosis and a foreign body reaction with endothelial

giant cells also is present. In a small percentage of the cases, the lesion is a sclerosing angioma.

Prognosis. The lesion reaches a definite size and then remains stationary.

Treatment. Excision with the scalpel is standard therapy if the patient is worried about the lesion.

NURSING ASPECTS

If benign tumors are to be removed surgically the patient who is nervous should be given a mild sedative 15 minutes before the operation is begun. The patient should be examined for the presence of keloids and questioned regarding sensitivity to procaine, and the heart and the blood pressure should be examined routinely.

Preparation. The area should be scrubbed with tincture of green soap then alcohol and a stainless antiseptic should be applied. After the area is draped properly the light is focused and sterile gloves are spread out for the operator a few encouraging remarks to the patient are helpful. Equipment includes sterile towels, 1 to 2 per cent procaine solution containing 1:1,000 epinephrine, Bard-Parker knives (11 and 15) flat and curved scissors, Hagedorn needle, fine-pointed and blunt-pointed tissue forceps single-hook retractors small hemostats, needle holder white silk, borschalt and fine catgut sutures, sterile dressings, Telfa non-adherent sterile strips, Gelfoam, liquid surgical dressing bandages and biopsy bottles properly labeled.

Malignant Growths of the Skin

ETIOLOGY OF CANCER OF THE SKIN	CARCINOMA (<i>Contd</i>)
THE PRECANCEROUS DERMATOSES	SQUAMOUS-CELL CANCER
CUTANEOUS HORNS	PAGET'S DISEASE OF THE SKIN
SENILE KERATOSES	BOWEN'S DERMATOSIS
SEBORRHEIC KERATOSES	MELANOMA
CHRONIC SOLAR DERMATITIS	SARCOMA
CARCINOMA	IDIOPATHIC MULTIPLE
BASAL-CELL CARCINOMA	HEMORRHAGIC SARCOMA
BASOSQUAMOUS CARCINOMA	METASTATIC SKIN CANCER
	PREVENTION OF SKIN CANCER
	NURSING ASPECTS

The malignant tumors of the skin are of great importance because of their serious and destructive nature

They are characterized by a steady invasive, infiltrative growth local destruction and in some types, a tendency to metastasize. The lesions consist of cancer cells which vary in size and shape, and in their invasive tendencies and possess irregular cellular outlines and large deeply staining nuclei.

Types of Cancer Cancer is classified according to its origin and growth. Epithelial growths are called carcinoma connective tissue (mesodermal) growths are called sarcoma. Primary growths are those which originate in the skin secondary or metastatic cancer originates internally

Cancer of the skin may be divided into the following groups.

CARCINOMAS (1) basal-cell (2) squamous-cell (3) transitional (basosquamous) (4) intermediate, (typical neither of basal-celled nor squamous-celled cancer and (5) melanocarcinoma.

SARCOMAS *Localized types* (1) round cell (2) large and small spindle-cell (3) giant cell (4) fibrosarcoma and (5) neurogenic sarcoma. *Generalized types* (1) generalized nonpigmented sarcoma (2) melanocarcinoma and (3) multiple lymphosarcoma.

ETIOLOGY OF CANCER OF THE SKIN

While the actual cause of malignancy is unknown, there are various predisposing and contributing causes of cutaneous cancer. One thing is fairly certain—every cancer starts from a pathologic condition or soil which may or may not be visible to the naked eye.

Predisposing factors in the causation of cancer

AGE. One must not be influenced for or against a diagnosis of cancer by the age of the patient. However generally speaking basal-cell carcinoma tends to occur in patients past 50 squamous-cell carcinoma in those under 50 years of age. As is well known sarcoma is primarily a disease of the young.

OCCUPATION. Those exposed to physical irritation produced by the carcinogenic rays of the sun to wind and to heat (farmers, sailors, and stokers) are predisposed. Chemical irritation from soot, creosote paraffin carcinogenic oils and tobacco is also a frequent precursor of malignancy.

RACE. Cancer of the skin is rare in the Negro, whose pigmented skin probably plays an important part in his protection.

TRAUMA when repeated over a period of time may initiate skin cancer. In some cases, squeezing blackheads, irritation of the bridge of the nose from eyeglasses and irritation of the mucosal flaps of the mouth from defective dental appliances appear to have influenced the development of cancer.

GEOGRAPHY. The incidence of basal-cell cancer is greatest in those states having year round sunshine.

TYPE OF SKIN. Those patients with seborrhoeic skins are predisposed to basal-cell cancer those with numerous senile keratoses, to squamous-cell carcinoma.

PRECANCEROUS LESIONS. The stimulating effect of syphilis on leukoplakia of trauma on pigmented nevus and of x rays on kraurosis vulvae is well known.

LOCATION. The mucous membranes and the mucocutaneous junctions and the backs of the hands are frequent sites for the development of squamous-cell cancer. Basal-cell cancer has a special predilection for the face although it may occur anywhere on the body.

HORMONES. The role of hormones in the production of cutaneous carcinoma is still under investigation.

CHRONIC INFECTIONS. Squamous-cell carcinoma may occur in old sinus tracts, in patches of lupus vulgaris and in old ulcers.

CELL RESTS. Cancer often arises from dermoid cysts and other nevi, including embryonic groups of cells in the walls of the hair follicles, the sebaceous or the sweat glands.

ARSENIC. Occupational cancer from external contact with arsenic compounds occasionally occurs. The triad, melanosis, keratosis and multiple superficial epitheliomatosis, may result from the internal administration of inorganic arsenic (e.g., Fowler's solution).

RAPIDITY OF GROWTH. Basal-cell cancer has a slower rate of growth than the squamous type, the ratio being about 1:4. A sudden increase in growth of a basal-cell lesion may mean transition from a basal-cell to a squamous-cell growth.

DEGREE OF MALIGNANCY. Broder's classification of the degree of malignancy, based on cellular differentiation, is not recognized generally by dermatologists, since the histology and the prognosis are not always related in skin cancer.

DEATH RATE. About 6 per cent of the deaths from cancer in the United States are attributable to cancer of the skin and the oral mucous membranes.

THE PRECANCEROUS DERMATOSES

A precancerous lesion is a phase between the normal and a cancer. Over 20 per cent of the precanceroses already show malignant changes. While they vary in their clinical pattern, they all have a common pathology, consisting of abundant mitoses, disorganization of the epithelial cells (dyskeratosis), anaplasia, acanthosis and a polymorphism of the cells and their nuclei.

Predisposing causes include the following: (1) physiologic involution of the skin; (2) chronic infections; (3) atrophic dermatoses; (4) old scars; (5) chemical irritants; (6) physical irritants; (7) malignant degeneration of benign tumors; and (8) repeated minor trauma.

CUTANEOUS HORNS

Cutaneous horns are hypertrophic horny growths arising from senile keratoses.

Clinical Characteristics. These lesions have characteristics of both warts and senile keratoses. Most cutaneous horns occur on the cheeks or the temples, but the penis and the dorsum of the hands are also sites.

The growths are usually single straight or curved. They are hard and dry and vary in color from light brown to black. Their size is variable some are only a few millimeters long, while others are several centimeters in length. The base of the lesion is slightly raised and is infiltrated.

Course. The lesion may drop off several times a year only to recur. About 12 per cent of cutaneous horns undergo malignant degeneration after several years (Leverth).

Pathology. The hyperkeratosis which is marked and out of all proportion to the acanthosis, produces a flattening of the rete pegs. The basal layer is intact. Prickle-cell carcinoma may develop in the lesion.

MICROSCOPIC EXAMINATION of the base of the lesion should be a routine procedure. If the following changes are found, malignancy has developed: (1) atypical epithelial proliferation, (2) dyskeratosis, (3) broken basal-cell membrane, (4) inflammatory reaction.

Treatment. Surgical excision is the standard treatment.

SENILE KERATOSES

Senile keratoses are very common in the elderly and practically always are associated with a senile skin. Their importance



FIG. 136. Cutaneous horn on site of senile keratosis.

lies in the fact that they are apt to degenerate into squamous-celled or basal-celled cancers.

Clinical Description. The lesions usually are found on the face and the backs of the hands, areas exposed to the actinic rays of the sun. If the lesions appear on the mucous membrane of the lower lip they are more apt to undergo malignant degeneration.



FIG. 137 Tumorlike keratoses of the hand (From Drs. A. B. Loveman and M. T. Fliegelman)

Senile keratoses are usually multiple, irregular in shape, dime-size to quarter size and yellow, gray or brown in color. They appear to be imbedded in the skin. The tumorlike types are rare.

They may be thickened, verrucous and horny or covered with adherent scales. The early lesions are usually flat. If the lesion becomes raised or if inflammatory signs appear, malignant changes probably have supervened.

Etiology. Prolonged exposure of a senile skin to the actinic rays of the sun is a predisposing factor.

Differential diagnosis is from the seborrheic type, seborrheic dermatitis and lupus erythematosus.

Pathology There is a marked hyperkeratosis, with the horny layer dipping into the follicles and the ducts. Acanthosis is exaggerated, with fingerlike processes extending into the corium, but the basal-cell membrane is intact unless malignancy has developed. The underlying corium is the seat of a mild inflammatory process.

Course. Squamous-cell cancer often develops in the lesions; occasionally basal-cell growths arise.

Treatment. When senile keratoses occur on the lips they should be destroyed immediately. In the aged, only the frankly malignant growths should be removed as they occur. Keratoses are best removed by electrodesiccation, although the cautery or surgical methods also may be used. Theoretically vitamin A should be useful in some cases. Ointments are ineffective.

Prophylaxis. The use of creams or oils helps to keep the skin soft and free of superficial keratoses.

SEBORRHEIC KERATOSES

These keratoses are softer and more superficial than the senile variety. They usually occur on the trunk rather than on the exposed surfaces and rarely undergo malignant transformation. A greasy skin, or seborrheic soil, is usually present, but the relation of these growths to seborrhea has not been proven.

Clinical Description. Seborrheic keratoses are frequently grouped or multiple. The face, the back, the sides of the trunk and the submammary areas in women are frequent sites. They appear to be "stuck on the skin" unlike the senile type and often are elevated considerably. The surface is smooth or verrucous. It rarely is scaly or crusted. The color varies from yellow to brownish black. Itching is a frequent complaint. The mucous membranes, the palms and the soles are not affected.

Varieties. (1) Hyperkeratotic, (2) acanthotic and (3) dyskeratotic.

Pathology. This depends upon the stage of the lesion. The horny layer, which is loosely attached, is moderately thickened and dips down into the mouth of the follicles. There is a marked irregular acanthosis which often is cut up by islands of epithelial cysts, giving it a netlike appearance.

Course. They are characterized by slow evolution. The hyperkeratotic types rarely give rise to basal-cell cancers.

Treatment. None is necessary unless the patient wishes the lesions removed for cosmetic reasons or for fear of malignant changes. Best results follow light fulgeration or freezing with ethyl chloride followed by curettage

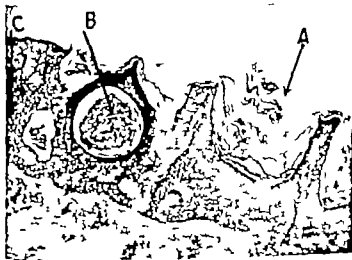


FIG. 136. Seborrheic keratosis, showing (A) marked hyperkeratosis, (B) epidermic pseudocyst and (C) moderate acanthosis

CITRONIC SOLAR DERMATITIS

Degenerative changes often develop in farmers, plainmen, ranchers, sailors and sportsmen who expose themselves to the actinic rays of the sun over a prolonged period.

The changes which occur are definitely precancerous and consist of pigmented macules, dilated capillaries and atrophic areas. The entire skin is leathery, freckled, pigmented, dry and shriveled with features of a chronic radiodermatitis, xeroderma pigmentosum and senile skin. When the lips are involved they are dry, scaly or fissured and keratoses are not infrequent. The face, the dorsum of the hands and the "V" of the neck are frequent sites.

Etiology The condition is common in those groups mentioned above. There is a regional incidence in the Southwestern states, where there is an abundance of sunshine throughout the year.

Blondes and red-haired individuals are more susceptible than those of other coloring. If malignancy occurs, it is usually a low grade squamous-cell variety.

Pathology When uncomplicated by the presence of keratosis the epidermis and the corium show the usual atrophic changes of senile degeneration.

Treatment. Prevention should be stressed. Farmers and others with this type of skin should have periodic examinations for cancer. The skin and the lips should be protected with ointments containing para aminobenzoate compounds.

CARCINOMA

Basal-cell Carcinoma

Basal-cell cancer which is relatively benign arises from the basal cells of the epidermis or the appendages of the skin (glands and hair follicles). Basal cells being rich in chromatin and having a high grade vital activity are more likely to be affected by stimuli



FIG 139 Multiple basal-cell epithelioma (rodent ulcers) in a man of 42 years of age. The diagnosis was proved histologically. (From Dr L. J. A. Loewenthal)

than the other layers of the skin. Pure types never involve the mucous membranes primarily. They are characterized by slow growth, the absence of metastasis, the presence of a waxy nodular edge and central ulceration or crusting. Cystic, pigmentary and squamous-cell changes may occur.

While single lesions are the rule, multiple lesions are by no means uncommon. See Figure 139.



FIG. 140. Rodent ulcer with invasion of the underlying structures. The lesion was destroyed with the actual cantery. Enucleation of the eyeball was necessary. The original lesion had been treated by an "arsenic paste" quack.

Location. This type of carcinoma has a special predilection for the face. About 70 per cent occur above a line extending from the junction of the upper and the middle third of the nose to the outer canthus of the eye. 25 per cent appear on other parts of the face. The remaining 5 per cent develop on other parts of the body.

Clinical Description. THE FIRST STAGE consists of the development of a flat plaque which gradually becomes depressed in the center. One or more mother-of-pearl nodules soon make their appearance in the border. These nodules are characteristically waxy, hard, nodular and semitranslucent. Running over

the nodular border which appears to be a defense mechanism are telangiectases resulting from pressure on the capillaries by the infiltrate in the corium.

IN THE SECOND STAGE the overlying skin breaks down and a shallow ulcer with a raised nodular waxy translucent border results. In some cases thin scarring occurs as the border advances.

THE THIRD STAGE is characterized by deep advancing ulceration and destruction of adjacent tissues (rodent ulcer)



FIG. 141 Amelanotic superficial epithelioma of 5 years' duration. The lesion was destroyed by electrodesiccation.

Clinical Varieties. Since basal-cell lesions change their characteristics with growth the following varieties are only temporary phases

THE MODULAR TYPE is the most characteristic. The typical nodules appear at some stage in the evolution of every basal-cell cancer. Sooner or later ulceration destroys them leaving crusts.

WARTY TYPE After growth is well established any of the types of basal-cell epithelioma may present a vegetating surface characterized by verrucous or fungoid changes.

THE CICATRIZING VARIETY consists of an irregular thin, flat scar which represents central healing surrounded by a nodular

border. This type usually occurs on the jaw, the forehead or the scalp. Nodules and ulcers may develop in the scar tissue under which the process may extend.

SUPERFICIAL EPITHELIOMATOSIS is a quiescent slow-growing lesion. It is characterized by single or multiple brown or dull-red, dry or moist, scaly or crusted areas of dermatitis and atrophy, surrounded by a delicate thin threadlike edge. Nodules may be present or absent. The disorder sometimes occurs in association with arsenical keratoses. This type must be distinguished from late syphilis, psoriasis and lupus erythematosus. Small lesions should be destroyed by fulgeration and large lesions should be removed by surgical excision with plastic repair if necessary.

THE MORPHIALIKE TYPE which is associated with dermal fibrosis, consists of a pink or yellowish waxy smooth, flat or depressed lesion surrounded by a very faint threadlike border. After a variable length of time ulceration occurs. The slow growth and the presence of dilated capillaries on the surface should awaken suspicion.*

THE RODENT ULCER (INVASIVE) VARIETY rapid in growth and nutritional requirements is characterized by extensive and mutilating destruction with involvement of the soft tissues or the bony structures.

THE PIGMENTED TYPE is a rare light-brown or dark brown lesion. It is usually nodular and is characterized by an increase in pigment in the basal layer which however does not affect the prognosis.

THE CYSTIC TYPE contains one or more clear opalescent cysts in the lesion which represent cystic degeneration of some of the masses of basal cells.

PATHOLOGIC TYPES. *The multicentric or multifocal variety* springs from several points of origin in the epidermis.

The adenoid type is cystic and shows that attempts have been made to reproduce the glandular structures and the hair follicles.

The intra-epidermic (psoriasiform) type may resemble the ordinary type of basal-cell lesion if the biopsy is taken at the edge of the lesion from the nodular border. However the central portions show intra-epidermal proliferation.

* See Caro, M. R. and Howell, J. B. *Morphea-like epitheliomas*, A.M.A. Arch. Dermat. & Syph. 63:53-60, 1951.



FIG. 142 Early basal-cell carcinoma of the cheek.

Some types of basal-cell cancers are congenital in origin and spring from foci in the appendages in an attempt to form fully formed glands or hair follicles or they may represent independent embryonic cell rests. Males are affected more often than females. While most of the cases are observed in patients over 50 they are observed sometimes in the 20-year-old to 40-year-old age group.

Pathology The typical basal-cell epithelioma starts as a downward budding from a very small group of basal cells in the epidermis or as a horizontal budding from similar cells in the pilosebaceous system. These islands, cords, strands or alveoli of proliferating cells are separated by an irregular hyperplasia of hyalinized connective tissue. A palisade of typical basal cells lines each island of cancer cells. These are columnar. They contain a large nucleus and little cytoplasm and take a deep stain. Cystic degeneration and hyaline degeneration are pathologic changes.

Diagnosis in the simple types is relatively easy. The following characteristics should be looked for:

- 1 Chronicity
- 2 No tendency toward spontaneous healing history of healing and crusting
- 3 Characteristic border—wavy shiny smooth, hard, translucent mother-of-pearl nodules in a rolled border
- 4 History of antecedent lesion or keratosis is found in some cases.

5 Biopsy (with adequate material) should be made in all cases. If the examination reveals granulation tissue a deeper section should be taken.

In order not to cause undue alarm, the term *basaloma* is preferable to *cancer* in discussing the diagnosis with the patient.

Differential Diagnosis. FROM ULCERONODULAR GUMMA by absence of a rolled pearly border absence of recurrences in the scar raw ham color more rapid evolution and therapeutic response



FIG. 143 Basal-cell carcinoma, showing (A) normal rete, (B) epidermal cyst and (C) cords of cancer cells springing from basal-cell layer extending downward into the corium.

FROM LUPUS ERYTHEMATOSUS which exhibits no nodules or threadlike border shows central atrophy follicular plugging and an adherent scale

FROM LUPUS VULGARIS which begins in childhood and contains apple jelly nodules.

SOLITARY MOLLUSCUM CONTAGIOSUM a SMOOTH VERRUCA VULGARIS and FOREIGN BODY TUMORS may simulate basal-cell cancers.

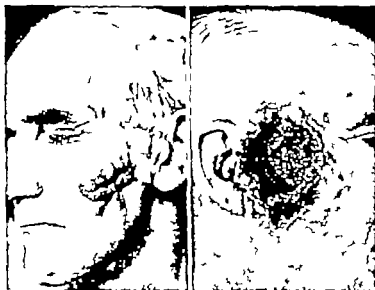


FIG. 144 (Left) Basal-cell carcinoma of the cheek (recurrent)

FIG. 145 (Right) Squamous-cell carcinoma (Dr McCuskey's cases)

It may be impossible to distinguish superficial epitheliomatosis from Bowen's disease or from extramammary Paget's disease without a biopsy.

Prognosis is usually good if the lesion is destroyed or removed in toto. Cosmetic results following treatment depend upon the extent and the depth of destruction. Since the lesions are radiosensitive and since the rate of growth is slow the percentage of cures is very high. Spontaneous cure is very rare. Neglected lesions may cause death from hemorrhage, septic pneumonia or meningitis by involvement of the deeper tissues or secondary infection.

Treatment. The type of therapy must be individualized and should depend upon the age of the patient, the location of the lesion, the clinical type, the type of previous therapy and the wishes of the patient. Surgical excision is indicated in lesions near vital structures and in cases in which cartilage or bone is involved, especially scalp, ear, hand and foot lesions and recurrent growths following radiation.

X-rays and radium in the hands of experts produce excellent cosmetic results if the lesion is in an area overlying sufficient subcutaneous tissue. The lesion should be curetted first to obtain a better view of its extent. X-ray dosage is given fractionally daily or every other day, in doses of 1,000 r for 4 doses if the lesion is under 5 mm. in thickness and 1,500 r for 3 to 5 doses if over 5 mm. in thickness.* If a basal-cell cancer does not disappear within three months of the beginning of radiotherapy it should be excised widely without delay. Electrodesiccation may be used to destroy small growths and those which do not lend themselves to surgical excision. The cautery is the standard treatment in dealing with the rodent ulcer type using the Mohs technique.

Recurrences are common in those cases complicated by scar tissue. In basosquamous types, in those previously irradiated, incompletely fulgerated or excised and in cases complicated by bone and cartilage involvement. The recurrent types should be excised widely with serial sections made to assure complete removal.

BASOSQUAMOUS CARCINOMA

These lesions are transitional, mixed or intermediate types and are caused by rapid growth in a basal-cell tumor resulting in a keratinizing cell degeneration. The basal-cell type of growth predominates but concentric lamellae or anaplastic cells are present.

Types. (1) **THE INTERMEDIATE-CELL TYPE** in which the pathologic cell is neither basal or prickle but has characteristics intermediate between the two forms.

(2) **THE MIXED TYPE** in which both basal-cell and squamous-cell structures are present.

Treatment and prognosis are similar to those for squamous-cell cancer.

SQUAMOUS-CELL CARCINOMA

Clinical Description. This variety (prickle-cell cancer) is characterized by rapid growth, atypical types and metastatic involvement. Histologically these growths demonstrate an attempt of the prickle cells to carry on their normal function

*For details of treatment according to location of lesion, see Effer J. J. and Effer W. D. *Carcinoma of the skin, therapeutic aspects*, New York State J. Med. 53: 2687 1953.

away from home (cornification) with the formation of "pearls" or cell nests.

The first indication of malignancy consists of a fissured warty growth or a flat, indurated area. This is followed by the formation of a circumscribed nodular lesion which often is concealed by a crust or an exudate but is not fixed to the underlying tissue. Due to rapid extension downward and laterally the blood supply of the lesion is choked off and an ulcer with a raw surface de-



FIG. 146 Squamous-cell carcinoma.
(From Dr. McCoskey)

velops. The ulcer has an indurated border. Usually it is everted. It bleeds easily and may be covered with a crust concealing a red granular base. With invasion of the underlying tissues, a cauliflower lesion finally results. All palpable lymph nodes should be subjected to biopsy as soon as discovered.

Etiology As a result of a more sheltered life and better care of the skin, this type of cancer is rare in the female. Males are affected more frequently in a ratio of 4 to 1 with the majority in the 50 to 70 age group. Squamous-cell cancer may develop from a traumatized nevus, an ulcer, a fissure, old scars or a precancerous dermatosis. A small number of squamous epitheliomas apparently arise *de novo* without any pre-existing lesion to account for them. Those occurring on the mucous membranes are the most malignant; those occurring on the skin are the least malignant.

The exposed areas of the skin are predisposed, the majority of the lesions occurring on scalp ears and dorsum of the hands. The mucous membranes most frequently involved are those of the mouth the tongue the lower lip and the mucous membranes of the penis and the vulva.

Pathology Hyperkeratosis is usually present especially in senile keratoses with malignant degeneration. There is marked hypertrophy of the prickle-cell layer with an irregular growth



FIG. 147. Squamous-cell carcinoma of the nose "button type."

of fingerlike projections downward and along the lymphatics. The prickle cells are large and polygonal and contain large nuclei and abundant mitoses. The cell nests (pearls) which are characteristic, consist of small groups of prickle cells surrounded by concentric rings of cornified cells which stain poorly.

Course. Metastasis to the regional lymph glands may occur early in the disease. If untreated the growth extends rapidly into the underlying cartilage bone and connective tissue, with metastasis to the viscera by blood-stream or lymphatic dissemination.

Prognosis. The outlook is serious. The ultimate outcome depends upon the early recognition of the condition. If the lesion is deep and if it is fixed to the underlying tissue and if metastasis to the region lymph nodes already has occurred the

chances for cure following removal are poor, and an ultimate fatal issue is the rule.

The value of grading in prognosis (Broder) according to the degree of anaplasia present in the section is controversial since the previous treatment, the location and the extent of the growth are also important factors.



FIG. 148 Squamous-cell carcinoma (Left) Of the lip (Right) On the tongue

Diagnosis. Accurate diagnosis is possible only by microscopic examination. Growths that are clinically basal-cell may show transitional squamous-cell degeneration when histologic sections are examined. Naturally the treatment is more radical in this type of case.

The general principles of cancer diagnosis are

1. Suspect cancer in solitary lesions in patients past 40.
2. Delay therapy until a definite diagnosis has been made by biopsy and a trained pathologist.
3. Be familiar with the precancerous dermatoses and the signs of malignant degeneration.

4 In case of doubt the patient should be referred to a tumor clinic in order to obtain the opinion of several experts.

Differential Diagnosis, BLASTOMYCOSIS. Squamous-cell carcinoma of the face and hands must be subject to careful study of the biopsy sections to rule out blastomycosis. **KERATOACANTHOMA** a benign epidermal tumor friable with a central keratin plug may be mistaken for squamous-cell cancer

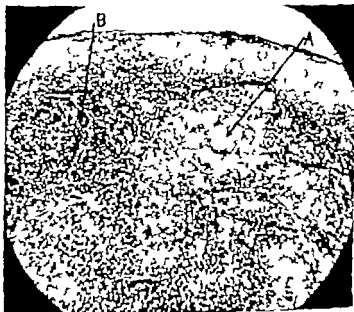


FIG. 149 Squamous-cell carcinoma characterized by (A) epibulb "pearls," and (B) projections of abnormal cells with hyperchromatic nuclei.

GRANULOMA INGUINALE TUBERCULOSIS VERRUCOSA CUTIS and VEGETATING PYODERMA may simulate squamous-cell carcinoma

The following features are suggestive in the differential diagnosis

	<i>Basal Cell Cancer</i>	<i>Squamous-Cell Cancer</i>
Age	Any age	Any age
Sites	Face	Mucous membranes and mucocutaneous borders. Scalp, ears and backs of hands

Surface	Depressed center Waxy rolled border. Very little inflammatory reaction. More or less crusting	Raised indurated lesion. Scaly inflammatory or ulcerating surface
Growth	Slow	Rapid
Pathology	Downward growth of strands or alveoli from basal-cell layer	Irregular growth of squamous-cells of prickle-cell layer with "pearl" formation

Treatment. The type of therapy used depends upon the duration, the extent and the location of the cancer and the presence or the absence of visible metastases. Lesions on the skin if not extensive, should be excised widely. Squamous-cell carcinoma of the lip without lymph-node involvement can be cured with 3,500 to 6,000 r x ray therapy in divided doses.* Radical excision and removal of the lymph nodes is indicated in the extensive growths. Invasive lesions and those involving cartilage are best destroyed with chemotherapy and microscopically controlled excision.†

PAGET'S DISEASE OF THE SKIN

Paget's disease is intraductal carcinoma involving the nipple and the areola of the breast. The condition is characterized by an eroded, crusted, weeping or eczematoid dermatitis with a sharply defined border associated with itching or tingling. The nipple may be normal, retracted, edematous or infiltrated. Beginning in the nipple or the areola of the breast, the process extends, after a variable length of time, to the surrounding skin.

Although the eczematoid stage may persist indefinitely malignant degeneration eventually supervenes, due to a breakdown of the symbiosis between the cancer cell and the skin.

Etiology The disease occurs in women over 40 years of age although males are affected occasionally. It seems that the Paget cell is a transitional type of basal cell originating in the basal-cell layer of the epidermis or the dermal appendages.

Pathology There is an underlying adenocarcinoma of the nipple in a large percentage of the cases. The characteristic changes are in the prickle-cell layer which contains Paget cells (large vacuolated edematous, pale-staining, often double-con-

*For details of therapy see Osborne, E. D. Treatment of malignant cutaneous lesions, J A M A 134:11, 1934

†See Mohs, F. E. Chemosurgery, A microscopically controlled method of cancer excision, Arch. Surgery 42:279 1941



FIG. 150 Paget's disease of the breast. Treatment consisted of a radical mastectomy with no recurrence after 5 years.



FIG. 151 Large Paget cells (Dr. L. V. Ackerson's case)

toured epithelial cells devoid of prickles, with large hyperchromatic nuclei) If epithelial proliferation has occurred, the pathology in the corium is that of a squamous-cell carcinoma.

Differential Diagnosis. This is mainly from seborrheic eczema, which occurs at any age is more inflammatory and pruritic, is less infiltrated is not progressive in character has an ill-defined border and responds to local therapy

Treatment is surgical and consists of a radical mastectomy

Bowen's Dermatosis

Bowen's dermatosis is a rare form of slow-growing intra-epidermal carcinoma characterized by dull-red, infiltrated, nodular or crusted patches. The mucous membranes may be involved also. The disease begins as a scaly or crusted nodule which later presents an eroded surface and spreads by peripheral extension. A rolled border rarely is present. Subjective symptoms usually are absent.

Pathology Like Paget's disease, the disease is carcinoma from the beginning although it has been classified with the precancerous dermatoses. A peculiar dyskeratosis is present in the prickle-cell layer. Once the disease invades the dermis it may assume the characteristics of any of the three types of carcinoma—basal basosquamous or squamous. Metastases are rare.

Differential diagnosis is principally from tuberculosis verrucosa cutis, *serpiginous* late syphiloderm and basal-cell carcinoma.

Treatment The lesions should be removed by surgical excision or surgical diathermy. Recurrences are rare after complete removal.

MELANOMA

Melanomas in the broad sense may be grouped into the benign types (pigmented nevi and freckles) and the rarer malignant melanoblastomas (melanocarcinoma and melanosarcoma). Malignant melanoma will be considered here.

Types of Malignant Melanoma. MELANOCARCINOMA (NEVO-CARCINOMA) These growths usually arise from a smooth hairless, flat, slate-blue or bluish-black nevus. As a result of trauma, irritation manipulation or unskillful irradiation the original growth may undergo a sudden or a gradual anaplasia consisting of increased growth tenderness and redness the development of

satellite lesions and a streaked radiating pigmentation. The draining lymph nodes are discrete and palpable.

Metastasis may occur at any time following malignant degeneration. Activation of a quiescent lesion may develop as long as 25 years after the first appearance of the growth.



FIG 152 Malignant melanoma, demonstrating extension of pigment beyond the limits of the original lesion (Dr McCuskey's case)

In spite of the fact that melanomas are the most malignant of tumors, certain types are clinically benign or inactive and are better left untreated. This type usually occurs in senile patients. Other varieties are rapidly malignant although they may spring from a similar lesion. Unfortunately it is impossible clinically or microscopically to foretell the prognosis when examining an activated melanoma. In some cases a definite diagnosis of malignant melanoma cannot be made by a study of the pathologic section.

NONPIGMENTED (AMELANOTIC) MELANOMA is a rare, yellowish-white stony hard tumor consisting of large polygonal cells which contain large nuclei undergoing mitosis. However melanin may be present in the biopsy section or in the metastases. They

are usually radioresistant, so that wide excision is the preferred therapy.

Etiology Malignant melanomata, which account for less than 1 per cent of all malignant tumors, may occur anywhere on the body but favor face feet subungual areas and extremities.



FIG 152A Malignant melanoma Infiltration of corium with atypical nevus cells (spindle and cuboidal) with partial disruption of epidermis due to upward pressure

According to Becker * 25 per cent of these lesions arise from flat, raised junction-type pigmented moles, and 75 per cent from lentigo maligna (acquired black macules). Repeated trauma is a factor (activation of quiescent black moles) in 75 per cent of the cases.

Pathology The nevus cell probably acts as a foreign body activating the benign melanoma into a destructive growth. Sections show nests of polyhedral round or spindle-shaped nevus cells with large hyperchromatic nuclei pigment granules in the epidermis and the corium and mitotic figures. The draining lymph

* Becker S W. Melanoma, diagnosis and treatment. A.M.A. Arch. Dermat. & Syph. 69: 11, 1954.



FIG 151A The general rule is that moles of the face are often benign (*left*) while those of the lower extremities are more likely to be malignant (From *Pfizer Spectrum* appearing in JAMA April 17 1954)

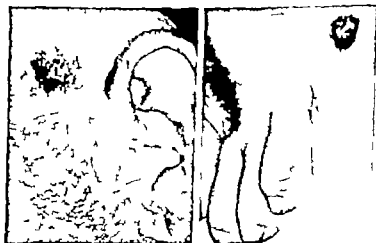


FIG 151B N rule is useful in melanoma except that all are potentially dangerous (*Left*) Malignant melanoma of the face (*Right*) Junctional nevus of foot, not now malignant but capable of becoming so (From *Pfizer Spectrum* appearing in JAMA April 17 1954)

vessels are packed with tumor cells. The predominating cellular type or structure bears no relation to the ultimate prognosis. In the nonpigmented type, no melanin is present, but the cellular structure of a nevus is retained.

Diagnosis. All suspicious lesions should be excised by scalpel surgery as early as possible. Microscopic diagnosis is so important that the grave responsibility of making a diagnosis should be borne by more than one expert pathologist. Most dermatologists can recall lesions erroneously diagnosed as pigmented nevi which eventually metastasized and proved fatal.

Tests for Melanin. In advanced cases the urine often contains melanin. If left standing the urine darkens, upon exposure, from a dark-brown to a black color. A few drops of ferric-chloride solution may be added to 10 cc. urine, with a resulting gray precipitate which turns black if melanin is present.

Differential Diagnosis. Often, the diagnosis of a melanoma can be made clinically, but in all cases the sections should be studied by an expert pathologist. Pigmented moles, pigmented seborrheic keratoses, blue nevus and pigmented basal-cell carcinoma resemble a melanoma clinically, but a biopsy and Perles' test for iron settle the question. Granuloma pyogenicum and ulcerated vascular nevi must be considered also.

Prognosis is very serious when the draining lymph nodes are palpable in lesions on the scalp and extremities and in those patients who have had inadequate therapy previously. Those cases which metastasize in the blood stream are rapidly fatal. Liver, brain and lungs bear the brunt of the metastases. Death usually occurs within one year after treatment regardless of the type of therapy used.

The prognosis of melanoma in children when treated by excision, is very favorable as a result of a hormonal inhibitory factor.

Prophylaxis. Pigmented moles should not be removed if they are not causing any trouble unless they are subject to irritation. The signs of beginning changes in a pigmented mole are as follows.

1. Increase in the size of the original mole.
2. Ulceration.
3. Hemorrhage.

4 Appearance of nodules or pigmentation in the skin around the original mole

5 Enlargement of the neighboring lymph nodes without changes in the original mole

Improper removal the use of caustics and unwarranted x-ray irradiation may transform a benign pigmented mole into a very malignant tumor

Treatment Malignant melanomata require immediate radical excision en masse of the primary lesion and the entire lymphatic drainage basin in one operation. X-ray therapy and radium are useless since less than 2 per cent of the lesions are radiosensitive (Stewart)

SARCOMA

Sarcomata are rare malignant mesodermal growths usually occurring in childhood and young adult life. They are radioresistant as a group and are characterized by invasive tendencies and recurrence after removal.

Type A biopsy is necessary to determine the pathologic nature of the growth.

MELANOSARCOMA is rare and springs from the blue nevus or the Mongolian spot.

NEUROGENIC SARCOMA sometimes occurs in von Recklinghausen's disease. It springs from the nerve sheaths and has a tendency to invade the bones.

SPINDLE-CELL SARCOMA consists of bundles or whorls of long, narrow cells and is not highly malignant

ROUND-CELL SARCOMA is a cutaneous or subcutaneous flesh-colored or bluish-red tumor consisting of small or large round cells. The lesions are highly malignant

RETICULUM-CELL SARCOMA consists of firm elevated reddish-brown nodules.

ANGIOSARCOMA is associated with proliferative blood-vessel changes and usually occurs on the face or the scalp.

FIBROSARCOMA (dermatofibrosarcoma protuberans) a reddish or purplish nodular tumor which is characterized by marked proliferation of the connective tissue, has a predilection for the thighs and the buttocks. It does not metastasize in spite of its growth characteristics.

LYMPHOSARCOMA usually occurs in the lymph nodes and rarely involves the skin. The cutaneous type consists of single or multi-

ple, flat or round, firm, painless, bluish-red tumors. Recurrence after removal is common and death invariably results from visceral metastases. See page 473

SECONDARY OR METASTATIC SARCOMA has the same features as the primary types.

Diagnosis. The clinical features of the sarcoma group vary considerably. In color they are nonpigmented red brown or purpuric. They vary in size from that of a pea to that of a grapefruit. Except for the Kaposi type, they have a tendency to occur in the young. A microscopic examination is indispensable for an accurate diagnosis.

Differential Diagnosis. The various types of lymphoblastoma may simulate sarcoma clinically. In these cases the expert dermatopathologist must be consulted.

Treatment. Wide excision followed by x ray therapy is the standard procedure. The lymph nodes in the area should be excised if they are involved. otherwise, irradiation is preferable. Inoperable cases often show temporary improvement from the administration of nuclear toxins.

IDIOPATHIC MULTIPLE HEMORRHAGIC SARCOMA

Kaposi's sarcoma consists of an eruption of bluish-red or mahogany brown solitary or multiple, verrucous, purpuric or translucent nodules, tumors or plaques. The primary lesion usually is described by the patient as a blood blister. The condition usually occurs on the lower extremities and the dorsum and the soles of the feet but the trunk may be involved also.

Visceral involvement may occur early in the disease but is often subclinical. Metastasis may occur to the gastro-intestinal tract, the liver the lungs, and the lymph nodes. The blood changes consist of a secondary anemia and a monocytosis. Bilateral edema of the legs is a characteristic feature.

Types. (1) The benign type consisting of relatively few lesions, and (2) the disseminated metastatic, which may be fatal.

Pathology. The inflammatory or early stage is characterized by dilatation of the capillaries and the lymph vessels. Iron pigment is present. The granulomatous stage consists of proliferation of newly formed blood vessels and numerous small hemorrhages. In the late or neoplastic stage the microscopic picture may be that of an angiosarcoma a fibrosarcoma or a spindle-cell sarcoma. The disease appears to spread along the lymphatics.

Course. The disease which runs a slow but progressive course is characterized by a low grade of malignancy.

Etiology. The cause is unknown. The disease has been considered to be a benign cellular reaction of the embryonic cells, a generalized hemangiomatosis or a manifestation of a reticulo-endotheliosis.



FIG. 154 Kaposi sarcoma (From Dr. Beatrice H. Kuhn)

Differential diagnosis is mainly from hemangioma with verrucous changes, hypertrophic lichen planus and melanosis.

Prognosis is poor. Death usually occurs within from 5 to 10 years.

Treatment. X-ray irradiation with collateral arsenic therapy is standard treatment for the early types. Wide excision followed by irradiation, is recommended for the advanced varieties. Nitro-

gen mustards often produce clinical remissions and general improvement.

METASTATIC SKIN CANCER

About 1 per cent of metastases from visceral carcinoma involve the skin. The scalp is a frequent site. The lesions are asymptomatic nodules and assume the histologic features of the primary growth. The prognosis is serious, of course. Diagnosis is by biopsy study of the sections. X-ray radiation or radium therapy is palliative only.

PREVENTION OF SKIN CANCER

The patient can avoid cancer of the skin by attention to proper hygiene, avoidance of excessive sun, securing annual examination of the mouth and the teeth, attention to proper fitting of glasses and by seeking expert advice regarding any sore scar birthmark or growth which takes on a change in size or appearance.

Physicians can prevent cancer by removing all suspicious keratoses, using care in prescribing inorganic arsenicals, insisting on biopsies on all suspicious growths and avoiding "piddling" with pigmented and precancerous lesions.

Selection, frequent inspection and protection of the worker in industries in which carcinogenic substances are used will reduce the incidence of industrial cancer.

Every persistent lesion should be considered in terms of cancer. Adequate biopsy material should be made available to the pathologist and all vague biopsy reports should be looked upon with distrust.

All tumors of the skin, of uncertain origin if subjected to repeated trauma, should be regarded as being potentially malignant and should be removed by wide surgical excision as a prophylactic measure.

NURSING ASPECTS

Preparations are similar to those for the removal of benign tumors except that squamous-cell carcinoma and melanoma usually require major surgery. If electrosurgery is indicated the surgical diathermy machine must be examined beforehand to make sure that it is in good working order and proper preparations should be made. Patients should be confined to the hospital for a few days after the operation to avoid such complications as infection, hemorrhage and reactions from treatment.

The Metabolic Dermatoses

CALCINOSIS CUTIS

NECROBIOSIS LIPOIDICA

DIABETICORUM

XANTHOMATOSIS

XANTHOMA PALPEBRARUM

NEVO-XANTHO-

ENDOTHELIOMA

JUVENILE XANTHOMATOSIS

XANTHOMA DIABETICORUM

DISSEMINATED XANTHOMA

TOSIS

XANTHOMA TUBEROSUM

HAND-SCHULLER-CHRISTIAN

DISEASE

THE AVITAMINOSES

VITAMIN A DEFICIENCY

THE AVITAMINOSES (*Contd.*)

VITAMIN B₁ DEFICIENCY

VITAMIN B₂ (O) DEFICIENCY

PYROXIDINE (VITAMIN B₆)

DEFICIENCY

B₁₂ DEFICIENCY

VITAMIN-C DEFICIENCY

VITAMIN-D DEFICIENCY

VITAMIN E DEFICIENCY

VITAMIN K DEFICIENCY

VITAMIN P DEFICIENCY

OTHER NUTRITIONAL

FACTORS

MULTIPLE VITAMIN

DEFICIENCY

NURSING ASPECTS

The normal functions of the skin depend upon a delicate balance between the physiochemical and the endocrine processes. While practically all disease is metabolic in origin, some of the rarer dermatoses are associated with definite disturbances of fat, carbohydrate, calcium, protein, mineral and water metabolism. These conditions are usually chronic and may be localized or systemic. The diagnosis is made by biopsy, special tissue staining, clinical and blood-chemistry findings.*

CALCINOSIS CUTIS

Calcinosis cutis is a rare condition characterized by a localized or a generalized deposition of free and combined calcium salts in the skin and the subcutaneous tissues.

*For a more detailed discussion of the subject, see Warner Kurt: *The Skin Manifestations of Internal Disorders*, chap. 29, Metabolic disorders, St. Louis, Moabey 1947.

Clinical Description. Three types have been described (1) the primary or idiopathic, (2) the secondary type which occurs in scleroderma, acrodermatitis chronica atrophicans and dermatomyositis, and (3) the metastatic type which develops in cases of osteomyelitis, parathyroid adenoma and hypervitaminosis D. The diffuse type often sets in with pain and stiffness in the affected parts. These symptoms may occur at irregular intervals. The skin lesions develop insidiously with the formation of shot-like subcutaneous nodules or plaques which are painless and freely movable. These masses of calcium soap ulcerate through the skin following trauma or irritation, and leave irregular sinuses. Most of the cases have been reported on the hips, the abdomen, the pelvic region and on the extremities.

Etiology. The exciting cause is unknown. Calcium deposits may occur in widespread collagen diseases of unknown etiology (dermatomyositis, Raynaud's disease and acrodermatitis atrophicans) in certain types of renal diseases and in some cases of destructive bone disease. An abnormal calcium-phosphorus metabolism causes a deposition of calcium salts in the presence of localized cellular disturbance.

Pathology. A deposition of calcium salts chiefly in the form of calcium carbonate, pushes the collagen bundles and the fat lobules apart and results in a foreign-body reaction. The process may be preceded by an acute vasospasm, resulting in a necrosis of tissue which attracts free calcium. The free calcium probably combines with neutral fats, forming white deposits of soap. The blood calcium, phosphates and phosphorus are not elevated except in the disseminated systemic types.

Prognosis. In children, the disease sometimes disappears spontaneously. In adults, the disseminated types are apt to be chronic.

Diagnosis is usually not difficult and is based on the history, biopsy studies, chemical analysis of the deposits and roentgenograms. X-ray examination of the affected tissues reveals radiopaque deposits lying in the deep tissues.

Treatment. It is difficult to evaluate therapy since some of the cases clear up spontaneously. The standard treatment consists of placing the patient on a ketogenic diet to increase the elimination of calcium and prescribing ammonium chloride to change the pH of the tissue fluids. Disodium phosphate also has been used for this purpose. Large calcium deposits should be removed sur-

gically ACTH and hydrocortisone have been useful in the types secondary to dermatomyositis.

NECROBIOSIS LIPOIDICA DIABETICORUM

This is a rare type of metabolic disorder usually localized to the anterior aspect of the legs associated with a hypercholesteremia and local vascular fatty and necrotic changes.



FIG. 155 *Necrobiosis lipoidica diabeticorum*. The atrophic plaques have violaceous borders and persist indefinitely.

Clinical Description. The condition begins as a well-defined, round or oval yellowish, firm glistening papule. After a variable period of time the lesion spreads peripherally to form a glazed scleroderma-like plaque. The older lesions often consist of ulcers which vary in size from that of a quarter to that of a silver dollar. The center of the plaque is usually depressed, atrophic

and yellowish with dilated capillaries running along the surface. The border is violaceous and irregular in its outline. There are no subjective symptoms. The anterior surfaces of the legs are the usual site, although the calves, the thighs, the ears, the wrists and the hands may be affected also. One or more lesions may be present. Glistening deposits of cholesterol and linear hemorrhagic areas have been reported in the ocular fundi.

Course. The disease is slowly progressive and persistent. After reaching a certain stage the lesions regress, with scar formation.

Etiology. The condition may appear at any age. Apparently females are predisposed. Trauma may be a factor in the localization of the disorder. Although the cause is unknown some cases are probably due to abnormal fat, others, to defective sugar metabolism.

Varieties. There are two types (1) the diabetic and (2) the nondiabetic. The diabetic types do not depend upon the severity of the diabetes but upon the associated hypercholesteremia. The nondiabetic types, in association with chronic arthritis and other chronic affections, frequently become diabetic.

Pathology. There is a disturbance of the fatty tissues with areas of necrosis and lipoid deposition. The collagen bundles show granular degeneration and occasionally deposits of lipoids (rust areas). The elastic tissue is absent in the infiltrated areas. The blood vessels in the affected area often show an obliterative endarteritis.

Diagnosis is made by the clinical appearance, biopsy studies, increased total lipids and fatty acids, the presence of hyperglycemia or hypercholesteremia. If the blood sugar is normal a glucose-tolerance test should be made.

Differential Diagnosis. Xanthoma, sarcoid, morphea and granuloma annulare are ruled out by the history and histologic examination.

Treatment. Carbon dioxide snow may be effective in the early cases. A diabetic regimen including insulin has no effect on those cases of diabetic origin. Diamox (Lederle) a nonmercurial diuretic, may hasten involution of the lesions. The dose is 5 mg. per Kg. bodyweight daily or every other day.

XANTHOMATOSIS

The xanthomas are a group of conditions which Weldman has named the "yellowing dermatoses." They are the result of a disturbance of lipid metabolism of unknown cause. Probably no one etiologic factor is at fault. Whatever produces the disturbance the threshold of the cells is lowered permitting a lipid infiltration. Xanthoma lesions appear in areas of dense connective tissue where the flow of lymph is retarded, thus promoting the precipitation of lipoids. Trauma is not a factor in the production of the lesions. Their color is due to the yellow blood pigment carotene or its derivative, xanthophyll.*

Clinical Investigation. Each case requires the following studies in order to determine the type the prognosis and the etiologic factors (1) biopsy (2) blood pressure (3) examination of the blood serum for gross lipemia (4) blood sugar cholesterol phospholipids and neutral fat determinations, (5) electrocardiograms for possible coronary artery disease and (6) liver function tests.

Prognosis in cutaneous xanthomatosis is dependent upon the degree of systemic involvement.

General therapy includes the elimination of all animal fats which contain readily assimilable cholesterol. Choline, methionine and other lipotropic substances are necessary in the secondary types with chronic liver disease

XANTHOMA PALPEBRARUM

These lesions are round or oval yellowish chamols-colored plaques, or infiltrations, which involve the upper and the lower lids. Women past 40 years of age are predisposed. This is the most common type of xanthoma lesion. The disorder may be associated with coronary disease arteriosclerosis and aortic disease. These may be present also in other types of xanthoma. Some investigators claim 40 to 70 per cent of these patients have elevated cholesterol readings, but we have not been able to substantiate this in our cases in which the disease was limited to the eyelids.

Treatment. Monochloroacetic or trichloroacetic acid cautiously applied are useful in destroying the growths. After the lesions are ringed with zinc-oxide ointment, the acid is rubbed vigorously

For a complete discussion of the subject, see Thannhauser S. J. *Lipidoses. Diseases of the Cellular Lipid Metabolism*, ed 2 New York, Oxford.



FIG. 135 A Xanthoma palpebrarum. The lesions were excised with a poor cosmetic result.

into the growths on the point of a sharp wooden applicator stick. The resulting crusts fall off within a week or 10 days. Re-treatment may be necessary. Excision, unless done skillfully is apt to cause ectropion. A low-cholesterol diet and liver studies are necessary in those cases with high-cholesterol and lipoprotein blood values.

NEVO-XANTHO-ENDOTHELIOMA

This is a rare eruption of yellowish-brown nodules, which generally makes its appearance soon after birth. Usually the extensor surfaces, face and the trunk are involved. These xanthomatous lesions are not nevi in the ordinary sense of the term nor are they malignant.

Diagnosis is made by prolonged observation and by histologic examination which reveals endothelial cells, Touton giant cells (the nucleus forms a complete circle) and foam cells (phagocytes containing fat). The blood lipoids are within normal limits.

Differential Diagnosis. Pigmented moles and urticaria pigmentosa must be excluded.

Spontaneous involution usually occurs within three years.

Treatment is unnecessary.

JUVENILE XANTHOMATOSIS

This condition which occurs in infants, is characterized by a generalized eruption of numerous smooth firm yellowish nodules. The eruption is apt to be thickest on the trunk. When the face is involved the lesions often have a pinkish cast. Nodular and exanthematic types have been described. Some cases are associated with Hand-Schüller-Christian disease. Cardiac and coronary complications have been reported. If the liver is involved, there is usually an associated hyperlipemia. A familial tendency is present in some of the cases. Differential diagnosis is from urticaria pigmentosa and nevo-xantho-endothelioma. This type may or may not clear up spontaneously.

Treatment consists of dietary control with limitation of animal fats, since vegetables sterols are not absorbed.

Roentgenograms of the skeletal system should be made annually for bony defects because some cases may be formes frustes types of Hand-Schüller-Christian disease.

XANTHOMA DIABETICORUM

This is an eruption of yellow nodules, plaques and tumors in severe or poorly treated diabetic patients, associated with a disturbance of the lipoid metabolism. At the outset the lesions may present more or less inflammation. The lesions involve the extensor surfaces the palms the soles and the buttocks. A diffuse yellowish pigmentation of the skin also may be present (xanthochromia). The blood shows a high-grade lipemia and hyperglycemia. The onset is more or less sudden.

Pathology reveals deposits of intracellular and intercellular lipoids, but foam cells are sparse.

Treatment. The nodules usually disappear after a course of insulin injections and the institution of a diabetic regimen, although relapses are common.

DISSEMINATED XANTHOMATOSIS

This consists of an eruption of round oval or elliptical small yellow nodules which have a preference for the flexures. Dilated capillaries are usually present on the surface of the lesions. This type differs from other varieties in the tendency to involve the mucous membranes of the mouth, the pharynx, and the larynx. Xanthochromia, jaundice, biliary cirrhosis and diabetes insipidus may be present. There is no increase in the blood lipoids.

Etiology The disease may be caused by central changes in the hypophysis or the tuber cinereum. Some of the cases reveal a familial tendency.

Pathology The lesions contain Touton giant cells, foam cells and young fibrous cells in the corium.

Treatment. Dietary measures, including a low fat diet, are of little value.

XANTHOMA TUBEROSUM

Xanthoma tuberosum is an eruption of multiple yellow fibrous nodules on the extensor surfaces of the extremities, the elbows and the knees. The lesions also may involve the tendons and the palmar striae. There is a marked increase in the total blood lipoids and cholesterol. Coronary aortic and myocardial involvement are present in over 25 per cent of the cases. In the extensive cases cirrhosis of the liver may occur resulting in jaundice. Vascular occlusion from involvement of the intima of the larger arteries is not uncommon.

Etiology The cause of the condition is "an error in metabolism," but apparently there is an endocrine factor in most of the cases.

Prognosis is guarded. Sudden death may occur in those cases with cardiac involvement. The fibrous lesions are not affected by any therapy including diet.

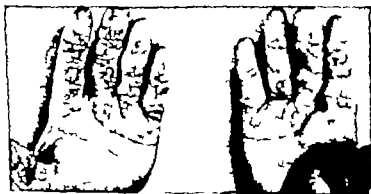


FIG. 156 Xanthoma tuberosum. Eruption consists of firm yellow nodules.

Treatment consists of a low-calorie diet, the avoidance of animal fats and thyroid medication if indicated. Surgical removal of the lesions results in recurrences at the site. Stress and strain should be avoided. Heparin in 25-mg doses I.M., weekly when given under laboratory control may reduce the blood cholesterol and reduce the size of the lesions. Inositol or a synergistic com-



FIG. 157 Xanthoma tuberosa. The tumors recurred following surgical removal.

bination (Methischo) in 1-Gm daily doses has not been effective in my experience.

HAND-SCHULLER CHRISTIAN DISEASE

Eosinophilic xanthomatous granuloma is a rare metabolic disturbance consisting of the following features: generalized xanthomatous-skin papules and nodules, areas of soreness over soft tissues, enlargement of the liver and the spleen, generalized adenopathy, purpura, diabetes insipidus, exophthalmos, hyper

ostotic defects in the membranous bones the skull the pelvis and the ribs, and arrested development. The disease is usually fatal when it occurs in children, although cases in adults are mild and recover spontaneously unless the disease becomes generalized.

Etiology The disease is considered to be a systemic reticulo-endothelial granulomatosis.

Differential diagnosis must be made from eosinophilic granuloma and Letterer Siwe's disease.

Pathology Sections from the skin lesions show the typical foam cells and reticulo-endothelial cells with vesicular nuclei.

Treatment. X ray therapy causes a partial or complete involution of the osseous lesions and may stimulate regeneration. Low fat diets are of no value. Steroid therapy may be useful.

THE AVITAMINOSES

Vitamins are necessary in cellular-enzyme reactions concerned with protein metabolism. When indicated they are of value but should not be prescribed indiscriminately and add to the cost of medical care.

The severe vitamin deficiencies that produce skin manifestations are not difficult to recognize (pellagra, scurvy etc.) but the mild types often pass unnoticed because they complicate other diseases or consist of nonspecific symptoms.

VITAMIN A DEFICIENCY (PHTHYRODERMA)

This is a common form of vitamin deficiency. Since vitamin A is necessary in normal epidermal metabolism, a deficiency results in altered epidermal structures. Xerophthalmia keratomalacia, night blindness, Bitot's spots photophobia dry harsh skin "goose skin" follicular keratoses, acneiform eruptions and dry brittle nails are included in the conditions caused by avitaminosis A.

Etiology Assimilation of vitamin A is greatly reduced in intestinal, biliary hepatic and pancreatic diseases in which prolonged faulty fat metabolism occurs.

Diagnosis. The therapeutic test with large doses of vitamin A is the only reliable one. Biophotometric and blood-concentration tests are of secondary importance.

Treatment consists of an adequate diet, containing milk, eggs and butter and the administration of concentrated vitamin A, for not over 2 months cod liver oil or haliver oil. Some cases show



FIG. 153. *Pityriasis rubra pilaris* with typical involvement of the palms. (From Dr. R. L. Howard)

a better response to carotene. For the toxic effects of vitamin A, see page 121.

Pityriasis Rubra Pilaris

This rare condition formerly known as lichen ruber acuminatus, has been classified with the scaly erythrodermias but the therapeutic response of some cases suggests that the disease may be a form of avitaminosis. The primary lesion is a hard yellowish-pink or reddish papule capped by a scale or containing a horny plug. Most of the lesions gradually become confluent and form thickened dry rough, scaly yellowish-red plaques. Associated lesions are diffuse scalliness of the scalp, seborrhealike lesions on the face, waxy hyperkeratosis of the palms and the soles and dystrophic nail changes. The general health is not affected.

Distribution is characteristic. Favorite sites are the backs of the hands and the fingers, especially the first and the second phalanges, the extensor surfaces of the wrists and the forearms, the anterior neck and the upper trunk. In late cases the disease becomes generalized.

Types (1) Juvenile (2) adult, (3) acute, (4) chronic.

Etiology The cause is unknown but vitamin-A deficiency has been suggested as being causative. Most of the cases occur in the 30-year-old to 50-year-old age group.

Pathology Hyperkeratosis, hyperkeratotic follicular plugs, with atrophy of the follicular wall, granulosis, slight acanthosis are present, with round-cell infiltration about the hair follicles and the sweat orifices.

Diagnosis is made by the presence of the characteristic follicular keratotic lesions on the backs of the fingers, the typical plaques and the biopsy sections.

Differential diagnosis is made from psoriasis, lichen planus, keratosis pilaris and ichthyosis.

Course. The disease is resistant to treatment and relapses are frequent.

Treatment consists of a high vitamin-A diet, vitamin-A medication (150,000 units daily) and 5 per cent salicylic acid in a petroleum base for local application. Concurrent cortisone or corticotropin therapy with vitamin A has been useful in some cases. Thyroid extract should not be administered concurrently since it destroys vitamin A. Some cases are refractory to vitamin therapy.

VITAMIN B₁ DEFICIENCY

Thiamin chloride has been suggested for the treatment of various diseases associated with neuritic symptoms because of its antineuritic properties. These diseases include leprosy, neuritis, herpes zoster and postzoster neuralgia. In general, the results with thiamin chloride have been disappointing.

Treatment consists of 10 to 500 mg of thiamin chloride daily by mouth, intramuscular or intravenous routes.

VITAMIN B₂ (G) DEFICIENCY

Acrodynia

Acrodynia is a syndrome in children characterized by various cutaneous and nervous symptoms. The hands and the feet are cold, clammy and edematous. The overlying skin has a characteristic pinkish hue. Severe itching is usually present.

A generalized macular, papular or purpuric eruption may be present. Fever, weakness, fatigue, paresthesias, calf tenderness, edema, muscular hypotonia, excessive perspiration, irritability, crying spells and upper respiratory infections are not uncommon. The disease may be related to pellagra. Some cases apparently

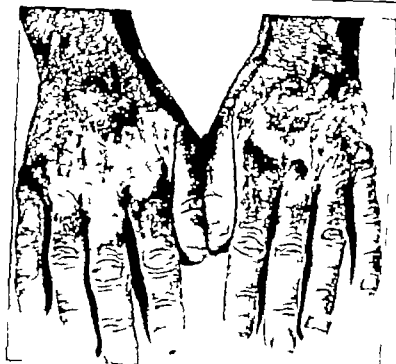


FIG. 159 Pellagra, demonstrating the characteristic involvement of the hands.

have been caused by mercurial intoxication. Edema and meningeal irritation have been found in the brain in fatal cases.

Treatment consists of liver fruit juices, yeast, cod-liver oil, thiamin chloride and nicotinic acid amide. BAL is effective in those cases with a history of exposure to mercury.

Pellagra

Pellagra is an acute, subacute or chronic, sporadic or endemic deficiency disease consisting of various degrees of cutaneous, mucous-membrane, gastro-intestinal and nervous involvement.

Mild cases frequently come to the attention of the dermatologist. These may consist of a symmetrical mild dermatitis with slight edema on the backs of the hands, a glossitis or dry cracked lips. Systemic symptoms may be lacking.

The characteristic dermatitis consists of a dull-red erythema affecting the face the anterior neck, the wrists, the backs of the hands, the ankles, the scrotum or the perineum. A scaly patch of erythema on the bridge of the nose, follicular hyperkeratoses of the nasolabial folds and a moderate alopecia are not uncommon. When the neck is involved the area of dermatitis is arranged in the form of a necklace or a collar. The dermatitis on the backs of the hands is sharply demarcated from the palmar surface and often extends up the extensor surfaces of the wrists and the forearms (areas exposed to light and irritation). After several weeks the erythema disappears and is replaced by pigmentation thickening and desquamation. Atrophy occurs in the late stages. Itching and burning are frequent in the early cases.

The mucous membranes are affected also. The lips are dry and fissured the tongue is red and smooth. Salivation is often present. Stomatitis is not uncommon.

The blood picture is that of a moderate to severe secondary anemia.

The systemic symptoms consist of diarrhea or constipation anorexia malaise, loss of weight, giddiness and irritability. Paralysis, neuritis and psychoses occur in the late cases.

Etiology The disease is believed to be a vitamin B-complex deficiency. The predisposing causes are (1) inadequate diet due to economic conditions, (2) dietary idiosyncrasies, (3) improper absorption of the vitamin due to functional or organic disease, (4) chronic alcoholism with limited diet.

The skin lesions are due to a photosensitivity resulting from the action of light on circulating porphyrins in the blood stream.

Pathology varies with the stage of the disease. In the chronic types there is hyperkeratosis, parakeratosis and an increase in pigment granules. The corium is the site of vascular dilatation, edema of the collagen bundles and some perivascular round-cell infiltration.

In the neurologic cases there is edema of the cerebral cortex, congestion of the posterior spinal-cord columns and inflammation of the posterior nerve roots and the spinal ganglia.

Course. The dermatitis recurs each spring and summer and fades gradually in the fall and the winter. The cutaneous and the general symptoms usually become more extensive and progressive with each succeeding attack. If untreated death may occur within five years from complications.

Diagnosis may be difficult in the absence of cutaneous symptoms which require intense sunlight for their activation. However a history of inadequate diet, gastro-intestinal and nervous symptoms, and glossitis or stomatitis should make one suspicious.

Differential diagnosis is made from acute lupus erythematosus, by the absence of the L.E. cells in the blood or bone marrow and solar dermatitis, which does not involve the covered parts.

Treatment. An adequate diet of 4 000 calories, including foods rich in vitamin B rest, good nursing care brewers yeast, liver extract.

THE DIET should be rich in peas, beans, fresh milk, meats, vegetables and fruits

NICOTINIC ACID AMIDE is specific for the dermatitis. It may be injected (1.5 mg. per Kg. body weight) or given orally (100 mg., three times daily) For children the dose is 10 mg. t.i.d. up to 6 years of age and 20 mg. from 6 years of age up to puberty. The drug is without influence on the polyneuritis, which must be controlled with thiamin chloride. Riboflavin is specific for the glossitis.

Plummer Vinson Syndrome

(See p. 571)

Ariboflavinosis

Ariboflavinosis consists of a magenta-colored smooth glossitis of the tongue fissures at the corners of the lips, increased vascularization about the limbus of the cornea, erythema and scaling of the acrotal sac and ill-defined superficial seborrhealike lesions on the scrotum, in the nasolabial folds and the nasal vestibules.

Etiology Most of the cases observed by the author occurred in the aged. The above signs are not specific for riboflavin deficiency but may occur in vitamin-A and vitamin-B and nutritional disturbances. Any prolonged nutritional metabolic or functional disorder interfering with the absorption and the metabolism of the vitamin may cause ariboflavinosis.

Treatment consists of large doses of riboflavin in conjunction with an adequate high-protein diet and rest. Large amounts of milk and vitamin A are useful.

NIACIN DEFICIENCY

This syndrome usually is associated with other B-vitamin disorders and is demonstrated best in pellagra. Subclinical types

present the following symptoms. Scarlet conjunctivitis and stomatitis, beefy red tongue, anorexia epigastric burning and organic-reactive psychoses.

PYROXIDINE DEFICIENCY (VITAMIN B₆)

While not a clearcut clinical entity cheilosis, angular stomatitis, seborrhealike lesions on the face and a hypochromatic anemia comprise this deficiency. The dose of the vitamin is 5 mg. t.i.d. or in multiple vitamin-B capsules as Becotla (Lilly).

B₁₂ DEFICIENCY

In many chronic dermatoses with macrocytic anemia, this vitamin, which is believed to be the maturation factor for the red blood cells, may be of value as supportive therapy. The vitamin is given by subcutaneous or intramuscular injection or orally.

VITAMIN-C DEFICIENCY

Scurvy is a deficiency disease characterized by bleeding and spongy gums, loosening of the teeth, purpuric eruptions and ecchymoses. Most of the cases seen in this country are subclinical. In infants the objective findings are unexplained hematuria and painful swellings over the long bones which appear on the x-ray plate as subperiosteal elevations (hemorrhages).

Some cases of acne vulgaris seem to improve on ascorbic acid although the effect of the vitamin on hyperkeratosis of the follicle is controversial.

The diagnosis is made by clinical findings therapeutic test with ascorbic acid, history of dietary deficiency the tourniquet test (lowered capillary resistance) and chemical assay of the blood (normal 0.8 per cent per 100 cc. of blood).

Treatment consists of orange, lemon, lime or tomato juice and ascorbic acid. In severe cases daily intravenous injections of 1,000 mg. ascorbic acid are used.

General tonics, oral hygiene and dietary supervision to prevent future recurrences are important.

VITAMIN D₂ DEFICIENCY

Avitaminosis D does not cause any specific dermatoses, according to our present knowledge. However vitamin D₂ (calciferol Driadol) has a pharmacodynamic effect in some cases of cutaneous tuberculosis parapsoriasis and sarcoidosis.

VITAMIN E DEFICIENCY

The significance of this vitamin in human nutrition has not yet been established definitely. Deficiency may produce degenerative changes in skeletal muscles, in the peripheral and the central nervous system and in collagen tissues. It is employed as natural mixed tocopherols or synthetic alpha tocopherol (from 25 to 50 mg., three times a week) in scleroderma, lupus erythematosus and in dermatomyositis, but the value of the vitamin in these conditions is controversial.

VITAMIN K DEFICIENCY

Purpura is the chief cutaneous manifestation resulting from hypoprothrombinemia. The causes of this deficiency are faulty utilization (hepatic disease), inadequate absorption (obstructive jaundice) or inadequate supply (newborn). The dose of Mena-dione U.S.P. is from 1 to 5 mg. given by mouth or by intramuscular injection.

VITAMIN P DEFICIENCY

Vitamin P (rutin or hesperidin) affects favorably the lowered capillary resistance found in some types of purpura. It is found in natural sources in lemon peel and orange peel. The dose is 50-100 mg. daily. Bleeding in hereditary telangiectasia may be controlled with daily doses of 50 to 500 mg. of rutin. Hesper-C (National Drug) a combination of hesperidin and ascorbic acid may be useful in peoriasis.

OTHER NUTRITIONAL FACTORS

Inositol, pantothenic acid and folic acid are used in chronic dermatoses with a nutritional factor as a complication. Choline which is essential in fat metabolism, may be beneficial in some types of xanthoma.

MULTIPLE VITAMIN DEFICIENCY

Multiple vitamins are employed in chronic eczemas, in the aged, resistant alopecias, nail disease and in chronic dermatoses where there is a nutritional factor.

NURSING ASPECTS

The nurse should be familiar with the various diets used in the metabolic and the vitamin-deficiency dermatoses. The recording of laboratory data, biopsy studies, the patient's weight and clinical data is an important phase of the nurse's duties.

The Atrophies of the Skin

SENILE ATROPHY

KRAUROSIS VULVAE

ACRODERMATITIS CHRONICA

ATROPHICANS

LINEAR ATROPHY

LICHEN SCLEROSIS ET

ATROPHICUS

CUSHING'S SYNDROME

ATROPHY OF SUBCUTANEOUS
FAT

HEMIATROPHY

AINHUM

SUBCUTANEOUS FAT NECROSIS
OF THE NEWBORN
NURSING ASPECTS

THE atrophies of the skin have been classified as either primary or secondary. These conditions may be generalized, localized or limited to certain tissues or appendages of the skin. The important feature histologically is the destruction of the fibrous and the elastic tissues, which causes atrophy. Clinically atrophic skin is thin, glistening, parchmentlike, wrinkled and white. There is often a slightly bluish-red cast, due to the reflection from the underlying veins. The loss of resilience results from the destruction of the elastic tissue which is characteristic of all atrophies.

The general causes of atrophy which are varied and complex, are (1) toxic, (2) inflammatory, (3) chemical, (4) interference with nerve supply, (5) physical (radiodermatitis, pressure, etc.), (6) interference with nutrition, (7) senility, (8) hormonal and (9) endocrine.

In general the prognosis for the atrophies is poor, the pathologic process being an irreversible one.

SENILE ATROPHY

This uniform generalized atrophy of the skin usually occurs after the age of 60. The skin is dry, thin, inelastic and wrinkled, with diminution of the subcutaneous fat. A fine branny scaling is present. On the face and the dorsum of the hands it is usual to find one or more pigmented atrophic and keratotic spots.

Pathology reveals a uniform atrophy of the epidermis with a loss of elastic tissue fragmentation of the fibrous tissue, atrophy of sweat glands, sebaceous glands, muscle fibers and hair follicles, with some increase in the pigment in the basal layer.

Complications. Localized eczematization neurodermatitis and nummular eczema are common in the winter months.

Prophylaxis consists of cleanliness and daily massage with soothing oils. The use of strong soaps, wool clothing home remedies or irritating applications should be avoided because of the danger of contact dermatitis or pruritus.

Treatment. A tub bath using Aveeno or a superfatted soap is permissible once or twice weekly. Two or more tablespoonfuls of Liquid Nivea may be added to the bath to avoid the drying effect. The skin should be anointed at bedtime with an oil satisfactory to the patient (liquid petroleum, Necto Lotion (Patch) Lubriderm (Abbott)).

Multiple vitamins to aid nutrition of the skin hormones if indicated and mild sedatives to lessen the pruritus, are important.

KRAUROSIS VULVAE

This is a degenerative condition of the mucous membranes of the vulva characterized by pruritus and atrophy of the surrounding parts. It frequently undergoes malignant changes.

Clinical Description. In many of the cases the disease is preceded by pruritus and a shiny dry red vulvitis. After a variable period narrowing of the vaginal orifice, atrophy of the mucous membranes and rigidity of the introitus occur. The mucous membranes become white glistening translucent and tense. Finally the entire introitus becomes shrunken with disappearance of the labia minora. Painful urination and dyspareunia are common symptoms in advanced cases. Eventually leukoplakic patches develop with or without secondary malignant degeneration.

Etiology. The cause of this disease is unknown. Predisposing diseases are endocrine dysfunction artificial menopause senile involution of the genitalia, chronic vulvovaginitis and chronic neurodermatitis.

Pathology. The main features are hyperkeratosis, atrophy of the rete with flattening of the rete pegs. Basophilic degeneration and homogenization of the collagen in the upper corium are characteristic.

Differential diagnosis. Lichen sclerosis et atrophicus, leukoplakia and pruritus with lichenification usually are distinguished on clinical grounds.

Treatment. While results are often disappointing, Vitamin A by mouth or injection may retard the progress of the condition.

Hunt* emphasizes the fact that (1) estrogens should not be used indefinitely in young women (2) estrogens have no effect on atrophic tissues and (3) any beneficial effects cease when the hormone is withdrawn.

Irritating vaginal discharges should be treated with alkaline douches perineal repair made if indicated and the fingernails kept short.

The pruritus may be controlled with Quotane ointment or 1 per cent phenol in lubricating jelly. Complete vulvectomy is advisable in chronic cases when leukoplakia or squamous-cell carcinoma (atypical proliferation of the rete downward into the corium) develops in the atrophic areas.

ACRODERMATITIS CHRONICA ATROPHICANS

This condition, which usually affects women of middle age, is a toxic process which affects the collagen tissues, resulting in diffuse permanent atrophy. In the majority of the cases the disease is limited to the upper and the lower extremities, but the buttocks and the lumbar region may be affected also. The disease consists of three progressive stages, all of which may occur in the same patient (1) the *inflammatory* or infiltrative, (2) the *atrophic* (3) the *scleroderma-like*. The onset is gradual with edema and redness. Symptoms of itching and burning may be present or absent. After several months the atrophic stage develops, characterized by the usual signs of atrophy. Later scleroderma-like changes may occur with the formation of fibrous nodules or trophic ulcers. Osseous changes involving the cortical layer of the long bones have been described. Arthritis may be associated with this condition but its relation is not known definitely.

Etiology is undetermined, but endocrines, varicose veins, cold, trauma and toxins have been considered as being possible predisposing causes. Over 70 per cent of the cases occur in Central European women, usually at the beginning of the menopause.

*Hunt Elizabeth Diseases Affecting the Vulva, p 221 ed 4 St. Louis, Mosby 1954.

Pathology consists of atrophy of the elastic and the collagen tissues, with perivascular infiltration vasculitis and atrophy of the subcutaneous fat.

Differential diagnosis should rule out purpura annularis telangiectoides, scleroderma with atrophy and poikiloderma vasculare atrophicum.



FIG. 160. *Acrodermatitis chronica atrophicans* in a 50-year-old woman, with extensive involvement of the buttocks, the thighs and the legs.

Treatment consists of massage with soothing oils, and a trial with estrogens in the chronic cases. Penicillin up to 10 million units, may be useful in the infiltrative stage but does not affect the atrophy.

LINEAR ATROPHY

Striae distensae occur on areas where mechanical, toxic, congenital or endocrine causes have ruptured the elastic tissue fibrils of the skin. The lesions are glistening white or purplish-gray slightly depressed parallel lines. When they occur during pregnancy they are known as *lineae albicantes*. Other causes are obesity and tumors and prolonged steroid therapy (adrenal androgenic action). They are not infrequent on the thighs, the hips, the buttocks and the breasts in obese women and adolescents.

LICHEN SCLEROSIS ET ATROPHICUS

This rare condition consists of a localized or a generalized eruption of flat or slightly raised, chalky white discrete or grouped papules which exhibit a central dark porelike depression. The usual localization in women is the vulva, the perineum, the anogenital area and the breasts. Although the disease may be confined to the vulva, the atrophy usually extends to the perineum and about the vulva in a "keyhole" distribution.

Etiology. The cause is unknown but the condition probably is caused by hormonal deficiency. Women of postmenopausal age are predisposed but the condition is sometimes observed in children.*

Pathology. Hyperkeratosis, keratotic plugging of the follicles, atrophy of the epidermis, with lymphedema and homogenization of the collagen in the upper corium without blood-vessel obliteration changes are significant.

Diagnosis is made on clinical grounds.

Differential Diagnosis. Kraurosis vulvae, vitiligo, the guttate type of morphea and senile genital atrophy must be considered.†

Prognosis is guarded. Spontaneous remissions are rare but the condition may wax and wane. Eventually the papules coalesce to form parchmentlike patches with hyperpigmented borders. When the vulvar or the anal mucous membranes are involved leukoplakia may occur as a complication.

✓**Treatment.** Some cases respond to injections of large doses of vitamin A (50,000 daily); others to estrogens. Anagen has been useful in selected cases. For topical use estrogen or vitamin A

*See Kindler T. Lichen sclerosus et atrophicus in the young, *Brit J Dermat* 65:269, 1961.

†See Wallace H J and Whitener I W. Vulvar atrophy and leukoplakia, *Brit J Dermat* 63:241-257, 1961.

ointments may be used or Quotane Ointment, if pruritus is present.

CUSHING'S SYNDROME

Cushing's syndrome is characterized by obesity of the trunk, the face and the neck while the extremities are usually normal. A puffiness of the malar pads gives the face a peculiar "moon-like" appearance. Other conditions may be present such as scanty menses, underdevelopment of the male genitalia, atrophy of the female breasts, hypertension and glycosuria. In a majority of the cases there is weakness, malaise and headaches. The cutaneous lesions consist of hirsutism (androgen stimulation), purpura, telangiectasia and symmetrical striae over the axillary folds, the trunk and the popliteal spaces.

Etiology. The syndrome may be caused by an oversupply of the glucocorticoid hormones of the adrenal cortex. Corticosteroid therapy may produce this syndrome when continued for longer than 2 months, but the symptoms disappear within a few weeks after treatment is discontinued.

Prognosis. The syndrome is chronic with spontaneous remissions. Death results from hypertensive cardiovascular disease, diabetes mellitus or intercurrent infections.

Treatment. X-ray therapy to the pituitary gland is the method of choice in cases without apparent adrenal involvement. Androgens reduce the edema and improve the nitrogen balance. A high-protein diet also is indicated. If the primary disease is in the adrenals subtotal adrenalectomy may be considered.

ATROPHY OF SUBCUTANEOUS FAT

This lipodystrophy is characterized by localized light-bluish atrophic areas. The affected parts appear sunken because of the underlying fat atrophy. The condition may be caused by toxins, injections of insulin or pituitary extracts and trophic disturbances.

Treatment is unavailing.

HEMIATROPHY

Hemiatrophy is a rare unilateral atrophy of the skin and the underlying tissues. It may be due to a nevus disorder, a postoperative symptom-complex resulting from injury to the autonomic nervous system or a trophoneurosis in syringomyelia, encephalitis, tabes and epilepsy. The type limited to the face is called

hemiatrophia faciei progressiva. There is shrinkage of the features and atrophy of the bony structures of the affected side.

Treatment is unsatisfactory

AINHUM

Ainhum ("to saw") is a rare atrophic disease of the toes usually occurring in male Negroes and characterized by a gradual development of a fibrous contracting band around the toes. The condition usually is limited to the small toe. After several months the distal portion of the toe becomes enlarged from pressure.

Etiology The disease is not confined to the tropics. It usually is the result of trauma from the use of ill-fitting shoes or from walking barefoot. The condition also has been described as a complication in anesthetic leprosy and plantar hyperkeratosis. Constricting and rarefying changes are found in the phalanx of the affected digit.

Differential Diagnosis. Congenital constricting bands and those associated with scleroderma and other atrophic conditions must be considered.

Treatment consists of cutting the constricting band or of amputating the digit if degenerative changes have occurred. Prophylaxis consists of wearing shoes or suitable footwear

SUBCUTANEOUS FAT NECROSIS OF THE NEWBORN

Adiponecrosis neonatorum is a rare condition of the deep tissues, characterized by bluish-red hard irregular nodular masses. The condition appears within three weeks of birth and disappears spontaneously within a year. Back, buttocks and extremities are favorite sites. Obstetrical trauma is a predisposing cause. The health of the infant is not affected.

Treatment is unnecessary

NURSING ASPECTS

The atrophies usually occur in patients past middle age. Since the skin in these conditions is thin, dry and easily traumatized, bathing should be infrequent, light mineral-oil ointments or vitamin-A ointments should be used frequently to control the pruritus and irritating ointments should be avoided. Alcohol rubs must not be permitted

Scleroderma and Allied Diseases

SCLERODERMA	PRETIBIAL MYXOMATOUS DEGENERATION
MORPHEA	SCLERODEMA ADULTORUM
DERMATOMYOSITIS	SCLEREMA NEONATORUM
MYXEDEMA	NURSING ASPECTS

SCLERODERMA

SCLERODERMA is a disease of unknown origin in which the affected skin the collagen and the subcutaneous tissues become hard and thickened with resulting atrophy. There are three types, a generalized or diffuse type called scleroderma, a form limited to the distal parts of the extremities (acroscclerosis) and a localized type called morphea.

The acute type begins gradually with the appearance of symmetrical areas of nonpitting edema, erythema or paresthesias affecting the hands or the feet. The overlying skin may be normal or erythematous in appearance. In children, the presence of joint symptoms may result in an erroneous diagnosis of acute rheumatic fever. The acute cases may terminate in spontaneous cure after a few months or they may enter the chronic phase.

The chronic type consists of two stages, an *infiltrative stage*, which is characterized by "tight" skin, increased rigidity and inflammation, and an *atrophic-end stage*. The *atrophic stage* occurs late in the disease and is characterized by atrophy of the skin and the bones. The face becomes masklike, wrinkles are obliterated, movements of the mouth are restricted and swallowing often is difficult. Due to fixation of the joints of the lower extremities, the patient has difficulty in walking. The fingers eventually become flexed as a result of fixation of the tissues (*sclerodactylia*). In many of the cases interference with nutrition results in ulcers over areas of pressure. The skin finally becomes pigmented or depigmented, alopecia is frequent and calcium deposits may occur.

Visceral lesions include involvement of the heart, the lungs and the gastro-intestinal tract.

Etiology The disease may occur at any age. Females are affected three times as frequently as males. At the present time the cause remains unknown. Scleroderma may be (1) the result of a functional disturbance of the vegetative nervous system since



FIG. 161 Progressive symmetrical scleroderma, with shrinkage and fixation of the nasal and the periorbital tissues. (From Drs. Orkison and Grindon)

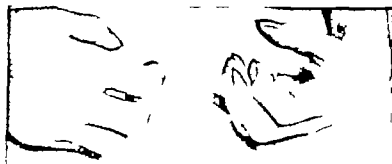


FIG. 162 Sclerodactylia. The joints are fixed, the tissues are "hardened," and the overlying skin is atrophic, smooth and shiny.

many of the cases begin with a Raynaudlike syndrome or (2) a disturbance of the hyaluronidase-hyaluronic acid system. There may be a predisposing history of acute infectious disease, trauma, syphilis or endocrine disorders, including hyperparathyroidism.

Differential Diagnosis. The acute types may be mistaken for acute lupus erythematosus or dermatomyositis. Sclerodactylia may be confused with Raynaud's disease, leprosy and syngomyelia. The atrophic stage should be differentiated from acrodermatitis atrophicans chronica.

Pathology. The disease probably starts as a vasospasm of the arterioles followed by edema of the collagenous bundles. The next phase is a gradual occlusion of the vessels, resulting in necrosis of the tissues. In the stage of infiltration there is hypertrophy with hyaline degeneration of the collagenous tissues and vascular dilatation. The stage of atrophy is characterized by a sclerosis of the connective tissue. Pulmonary, esophageal and myocardial fibrosis may occur in the chronic types. In sclerodactylia spontaneous absorption of the terminal phalangeal bones is not uncommon.

Prognosis. In most of the cases is usually good as to life expectancy. The outlook depends upon the extent of the involvement, the duration of the disease and the response to treatment. Occasionally recovery occurs without treatment. Death usually is due to intercurrent infection.

Treatment. It must be emphasized that some cases undergo spontaneous involution; that treatment must be given during the

edematous stage in order to have any beneficial effect and that some investigators have little faith in any type of therapy in this disease. The principles of treatment are as follows:

- 1 Keep the skin as soft and pliable as possible by massage with cod-liver-oil ointments by using hot baths or heat. Benadryl seems to have a beneficial effect in selected cases when given by mouth or injection.

- 2 Regenerate collagen and muscular tissue. The tocopherols (Vitamin E) and Chloroquine (Aralen) may be of some value.

- 3 Increase the excretion of calcium from the tissues by producing an acidosis (ammonium chloride) or by stimulating calcium metabolism (thyroid extract) or by controlling its distribution (dihydrotachysterol)

- 4 Overcome muscular weakness by using neostigmine bromide (from 15 to 30 mg., three times daily) and a high-protein diet.

- 5 Attempt prolonged vasodilatation with Meecholyt Ionto-phoresis, Meecholyt Bromide, tetranitrate.

- 6 As a last resort, remove vascular spasm by surgical measures, such as sympathectomy.

- 7 Remove infected teeth as being a possible source of infection. Avoid cold, tobacco and emotional stress.

- 8 ACTH, cortisone or Meticorten may influence the water metabolism of the collagenous tissues and produce temporary remissions.*

MORPHEA

The onset of morphea (localized type) is gradual and insidious, with the appearance of dime-sized patches. These are hyperemic at first but later assume an ivory white color. Well developed lesions have a smooth keloidal-like surface and are hard and thick. They cannot be wrinkled and appear to be attached to the underlying skin (hidebound). The *linear* type, which is saberlike or bandlike, may occur along the long axis of an extremity or along a nerve trunk, or it may involve one side of the face or the forehead. An associated hemiatrophy may be present in those cases where the face is involved. The *plaques* variety, or common type, consists of discrete hard, smooth, thick, flat, elevated or depressed patches, surrounded by a violaceous halo. The surface of the lesion is covered with follicular plugs and

*For an appraisal of therapeutic measures, see Evans, J. A. et al., Treatment of diffuse progressive scleroderma, J.A.M.A. 151:891, 1953.

plexuses of fine blood vessels. Common sites include the breasts, the abdomen and the thighs. Muscular atrophy of the affected areas may occur in rare cases.

Etiology The cause is obscure. In a few cases there is a history of trauma. In the linear cases which appear to follow nerve trunks, the old theory of trophoneurosis appears tenable until something better is advanced.

Pathology Pressure from below causes a thinning of the rete ridges and the epidermis. The changes in the corium consists of hypertrophy of the collagenous tissue, edema of the capillary walls and a perivascular lymphocytic infiltrate. In old cases sclerosis of the fibrous tissue, atrophy of the dermal appendages and the elastic tissue and obliteration of the blood vessels are present.

Differential Diagnosis Lipodystrophy atrophic lichen planus and the morphealike basal-cell carcinoma rarely confuse the experienced examiner.

Prognosis is guarded. The plaques may gradually disappear spontaneously, atrophy may result, or the process may remain stationary for an indefinite period.



FIG. 163 Morphea. The plaque was hard, smooth and elevated. Duration 2 years.

Treatment. There is no specific therapy. Artane has been advocated by Robinson, but the drug is too toxic for routine therapy. In most cases, 150 units of hyaluronidase injected daily into the center of the lesion for 10 days will be followed by perceptible softening of the lesion. The course may be repeated in a month. Daily massage with light mineral oil will decrease the sensation of tension. Thyroid or niacin therapy were not beneficial in our cases.

Hydrocortisone therapy is often beneficial especially in the multiple cases when continued for a month or longer. The usual precautions must be observed.

In the linear type causing deformity plastic surgery should be considered.

DERMATOMYOSITIS

This is a rare subacute or chronic inflammatory syndrome characterized by a gradual onset with nonpitting edema of the upper and the lower eyelids, nonspecific dermatitis and progressive multiple myositis. There is considerable diversity in the clinical picture.

In 8 per cent of the early cases the onset is characterized by a lupus erythematosuslike eruption of the face and heliotrope discoloration of the lids. The general symptoms consist of low irregular fever, progressive weakness, vague pains (muscle spasm), angina, difficulty in respiration and deglutition. Enlargement of the spleen is an early sign. Atrophy of the shoulder-girdle muscles may occur in the chronic cases. Bone lesions including osteocysts and osteoporosis, have been reported also. The associated cutaneous lesions consist of erythema, erythema multiforme or erythema nodosum. In many cases the onset of the disease is similar to that of early scleroderma.

Etiology. The cause is unknown but the following theories are held: (1) infection (2) light sensitization (3) vitamin-E or gonadal deficiency (4) disturbances of the hyaluronidase-enzyme system, (5) toxic reaction from focus of visceral carcinoma.

Pathology. In most of the cases the creatin of the urine is increased due to muscle injury. In about 20 per cent of the cases, biopsy will reveal parenchymatous degeneration of the muscles and separation of the myofibrils with perivascular infiltration. Examination of the muscular bundles reveals a lymphocytic in-

Treatment. Thyroid extract (fresh) in graduated doses (continued during the lifetime of the patient) potassium iodide Lugol's solution parenteral vitamins a high-protein, low-carbohydrate diet and massage are important.

PRETIBIAL MYXOMATOUS DEGENERATION

Localized, or Circumscribed, Type (Pretibial Myxomatous Degeneration) Usually the anterior aspect of the legs is affected. The lesions consist of hard, raised yellowish, waxy or rose-colored plaques resembling psoriasis. Verrucous, plaque, tubercle and papular types have been described. Many of the patients show no evidence of hypothyroidism. Although the majority of the cases follow subtotal thyroidectomies, the exact cause is unknown. The unopposed action of the thyrotropic hormone of the pituitary may be a factor. It appears to be unrelated to the general form of myxedema or to the persistence of the thyrotoxicosis.

Pathology is chiefly in the lower corium which reveals large amounts of mucin, an increase in fibroblasts and newly formed collagen tissue.

Prognosis poor for immediate results from therapy. Spontaneous involution occurs after 5 years.

Treatment. Temporary improvement may follow injections of hyaluronidase into the lesions and pressure dressings. Acetazola-mide (Diamox) 250 mg. B.I.D. combined with hydrocortisone may be effective.

SCLEREDEMA ADULTORUM

This is a rare disease which is characterized by a sudden onset of solid edema of the subcutaneous tissues. It usually begins on the head and the neck and spreads rapidly to other parts. In most cases there is complete resolution within a few weeks or months. As a rule there are no sequelae. The cause is unknown, although many cases follow a sore throat or an upper-respiratory infection.

The pathology consists of an edematous swelling of the collagen bundles.

✓ **Differential diagnosis** is from dermatomyositis, scleroderma and trichinosis.

Treatment is unsatisfactory but antibiotics, ACTH and fever therapy may prove to be satisfactory.

SCLEREMA NEONATORUM

Sclerema neonatorum is a progressive hardening of the subcutaneous fat in infants, often resulting fatally. The skin is tense, waxlike and frozen in appearance, the breathing is slow and difficult, and suckling is impossible.

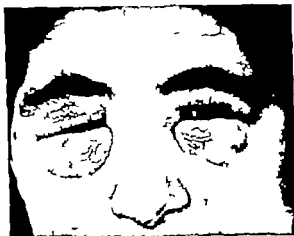


FIG 165 Sclerema neonatorum (Dr Bert L. Vallee's case)

Etiology Premature and feeble infants with subnormal temperatures, dehydration poor peripheral circulation or shock are predisposed. The condition probably is caused by a replacement of the olein of the normal fatty tissues with palmitin and stearin, resulting in changes in the melting point of the fat.

Pathology Atrophy of the adipose tissue with degeneration of the connective tissue trabeculae are the usual features.

Prognosis. The mortality rate is over 75 per cent.

Differential diagnosis is mainly from subcutaneous fat necrosis

Treatment consists of incubation care forced feedings to keep up the nutrition and friction to restore the circulation. ACTH or cortisone therapy if used early may be life saving.

NURSING ASPECTS

Patients with scleroderma often are hospitalized for a complete

physical examination, endocrine and special cardiac x ray and tissue studies blood chemistry studies and observation of the effect of corticotropin and vasodilator drug therapy. The skin should be massaged daily with nutrient oils. In advanced cases, the patient may require special care if the the general stiffness of the hands and the extremities interferes with feeding and rest.

Sclerema and edema neonatorum are treated in pediatric wards. The nurse should be familiar with the care of infants in incubators, the administration of fluids and general pediatric technique.

The Peripheral Vascular Diseases of the Skin

PURPURIC VASCULAR DISEASES
PROGRESSIVE PIGMENTARY
DERMATOSIS
ANGIOMA SERPIGINOSUM
PURPURA ANNULARIS
TELANGIECTOSES
REDGORES
VASOMOTOR NEUROSES
SYMMETRIC GANGRENE OF
THE EXTREMITIES

THROMBO-ANGIITIS
OBLITERANS
SICKLE-CELL ANEMIA
ERYTHROCYANOSIS CRURUM
ARTERIAL OBSTRUCTION
VENOUS OBSTRUCTION
VARICOSE VEIN COMPLEX
LIVIDO RETICULARIS
TELANGIECTASIA
NURSING ASPECTS

The blood vessels in the skin are necessary for control of local blood flow regulation of the blood pressure, nutrition, temperature control and tissue repair. Their size and number vary in different parts of the body. As far as the peripheral vascular system is concerned the arterioles are the most important factors in regulating blood flow. In order for the skin to function normally the arterial tone must be maintained. This is regulated by the vasomotor center via the sympathetic nervous system. The veins that lie in close proximity to the arterioles form a subpapillary venous plexus which probably acts as an important blood reservoir*.

The normal peripheral blood flow may be affected by various complex factors giving rise to changes in the color, the temperature and the nutrition of the skin. As a result the following clinical signs may develop: stasis, edema, cyanosis, livedo, purpura, ecchymosis and telangiectasis.

Etiology. Peripheral vascular disease may be caused by various known and unknown factors.

*For a discussion of the anatomy and the physiology of the peripheral vascular system, see Abramson, D. I. *Vascular Responses in the Extremities of Man in Health and Disease*, Chicago, Univ. Chicago Press, 1944.

INTERNAL Hormonal psychosomatic, abnormal mental states, toxins (tobacco drugs, etc.) physiologic states (pregnancy menopause and menstruation) cardiac and hepatic disease endocrine blood dyscrasias and disturbances of the autonomic nervous system.

EXTERNAL trauma, pressure chronic exposure to heat and cold.

Diseases of the veins usually affect the lower extremities and are produced by valvular defects, stasis or thrombi trauma and general and local causes of venous congestion.

PURPURIC VASCULAR DISEASES

The dermatoses in this group are possibly cutaneous manifestations of the same process, differing morphologically only in degree *

Randall and his co-workers found a pattern of increased capillary fragility and permeability with dilatation and proliferation of the capillaries in all of these dermatoses. No definite cause can be found although some cases appear to be unusual reactions to a stasis syndrome.

PROGRESSIVE PIGMENTARY DERMATOSIS

Progressive pigmentary dermatosis (Schamberg) is a type of stasis dermatitis consisting of cayenne-pepperlike punctiform lesions patterned in sharply defined maplike or crescentic patches. Usually the legs are affected. The disease appears first on the ankles and spreads upward along the lower extremities. During involution brown or yellow staining usually appears.

Section shows hemosiderin around the sweat glands, with dense inflammation of the corium and dilated blood and lymph spaces.

Treatment is usually unsatisfactory

ANGIOMA SERPIGINOSUM

Angioma serpiginosum is a rare nevold type of telangiectasia consisting of bright red punctate or annular lesions, areas of diffuse redness vascular papules and an irregular vascular network. The condition appears first on the extremities as red macules with central clearing. It tends to become generalized and is usually symmetrical. Pigmented and atrophic lesions may be present also. Although the disorder is usually stationary it may clear up spontaneously

*Randall, S. J. et al. Pigmented purpuric eruptions, Arch. Dermat. & Syph. 64:177 1951

Section shows no deposits of hemosiderin but necrosis of the basal-cell layer and dilatation of the blood vessels are frequent findings.

Differential diagnosis is from purpura annularis telangiectodes in which there is no history of a primary angiomatous lesion pigmentation and atrophy are present.

Treatment is unsatisfactory although erythema doses of ultraviolet light may benefit some cases.

PURPURA ANNULARIS TELANGIECTODES

Majocchi's disease is also a rare condition usually affecting young adults. Practically always it involves the lower extremities. The primary lesion is a bright-red pin-point macule. The eruption consists of telangiectasia, purpuric pigmented and atrophic lesions. The discoid and circinate patches tend to spread peripherally with the development of central atrophy. There is usually loss of hair in the involved areas. During the course of the disease there are periods of exacerbations and remissions, but the condition is not progressive.

Diagnosis is made by the clinical features, biopsy and prolonged observation. Many cases so diagnosed often are atypical simple purpuric eruptions.

Section shows hyaline degeneration of the blood vessels, miliary aneurysms and pigmentation. The disease usually clears up spontaneously after several months.

Differential Diagnosis. Purpura, radiodermatitis and early acrodermatitis chronica atrophicans must be considered.

Treatment consists of the use of vitamin C rutin and calcium therapy. The corticosteroids may produce temporary improvement.

VASOMOTOR NEUROSES

(Vasospastic Diseases)

SYMMETRIC GANGRENE OF THE EXTREMITIES

(RAYNAUD'S DISEASE)

Raynaud's disease is a vascular syndrome of unknown origin usually affecting the fingers and the toes, which, after causing local asphyxia often lead to necrosis.

Clinical Description. The onset is gradual. The first sign is usually edema, stiffness and numbness in the fingers. In most

cases there is a characteristic syndrome, consisting of syncope (blanching) asphyxia (cyanosis) and hyperemia. While the disease often is limited to the fingers, the tip of the nose and the ears may be affected also. After a variable length of time the finger tips become club-shaped, and nail dystrophies occur. Pain may be present or absent. In the final stage bullae, ulceration

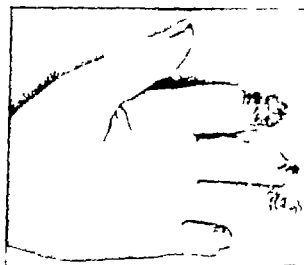


FIG. 166 Raynaud's disease with ulceration of the fingertips

and gangrene make their appearance. The disease is usually symmetrical.

Complications. Amblyopia hemoglobinuria epileptiform attacks and abdominal pain are not uncommon. Occasionally patients with this disease develop scleroderma or calcinosis.

Course. Recurrent attacks over a long period are common. In some cases the disease is mild and is limited to the fingers. Gangrene appears eventually as a terminal reaction.

Etiology. The disease is limited practically to the female sex. It usually makes its appearance in the second or the third decade. At the present time little more can be said concerning its causation except that it probably is a psychoneurosis. Predisposing causes are sudden shock, fright exposure to cold and pregnancy.

Prognosis is poor. A small percentage of cases recover completely. Tissue destruction is gradual but progressive.

Diagnosis. The history and the sex of the patient and the characteristic syndrome of syncope asphyxia and hyperemia are characteristic. In doubtful cases biopsy study of the capillary loops and oscillographic readings are helpful. Radioactive sodium may be used to determine the status of the peripheral circulation, employing the Geiger counter.

Treatment. Bed rest during exacerbations is advisable. Protection from cold is also important. Analgesics should be prescribed for pain. For local use, 2 per cent glyceryl trinitrate in lanolin may be beneficial. Good results from estrogenic substances have been reported. Mecholyl iontophoresis and suction pressure (parox) treatments are useful procedures. In advanced cases sympathectomy is a last resort.

THROMBO-ANGITIS OBLITERANS

(BUERGER'S DISEASE)

Buerger's disease is a chronic inflammatory disease of the vascular system of the extremities, limited to the male sex and characterized by severe pain vasomotor and trophic changes with terminal gangrene.

Clinical Description. The early manifestations of the disease are cramps or pain in the muscles of the calf the arch of the foot or the popliteal spaces. The pain is aggravated by exercise. The affected parts are hyperemic when dependent, pale when elevated. Pulsation of the dorsalis pedis artery becomes imperceptible. In the late stages, agonizing pain is present and ulceration and gangrene develop.

Pathogenesis. The disease is the result of a gradual occlusion of the blood vessels by thrombi.

Course is progressive and chronic. Sudden death may occur from cardiac or visceral thrombi.

Etiology. The disease is limited to the male sex, the majority of the cases occurring in the 25-year-old to 50-year-old age group. A large proportion of the patients are Jews. Among the predisposing causes are tobacco, exposure to cold and typhus fever.

Differential diagnosis is from Raynaud's disease arteriosclerosis and acrodactylia.

Treatment. The general treatment includes bed rest, care of the feet and protection from cold. Unless the patient gives up

smoking therapeutic measures are of no avail. Vasodilator drugs including papaverine and aminophylline are useful in the early stages. Contrast baths Buerger's postural exercises, thermostatic heat devices, rocking beds medical diathermy are beneficial at one time or another. Intravenous injections of sodium citrate and hypertonic salt solutions are of distinct value. Foreign protein therapy including typhoid vaccine produces a peripheral vasodilatation and relieves the pain indirectly. Peripheral nerve block is indicated to control the agonizing pain. In severe cases amputation or sympathetic ganglionectomy is a last resort.

Prophylaxis consists of avoidance of excessive exercise, elimination of tobacco, use of warm footgear to avoid frostbite, and proper care of the feet.

SICKLE CELL ANEMIA

This is a rare condition in which erythrocytes have a tendency to assume a sickle shape resulting in hemolytic anemia and chronic ulcers of the shins and the ankles.

Etiology The syndrome is confined to Negroes and those having Negro blood in their ancestry. About 8 per cent of Negroes have the sickling trait but only 0.2 per cent develop the anemia. The condition is familial and hereditary. It may occur at any period in life but is most common in children (Magner). Abnormal hemoglobin may be a factor.

Symptoms include weakness, abdominal pain, pains in the joints and the muscles, pallor of the mucous membranes, enlargement of the liver and spleen and chronic punched-out ulcers on the legs or the ankles.

Diagnosis. Sickling of the red blood cells is best observed by studies of sealed wet films. The degree of anemia present depends on the activity of the hemolytic process.

Treatment. ACTH and cortisone therapy has been found to be valuable by recent investigators.*

ACROCYANOSIS

(See p. 278)

ERYTHROCYANOSIS CRURUM

This is more common in England than in the United States and is associated with extreme weather conditions, which inter

*Rice, S. W. Healing of chronic sickle-cell leg ulcers with cortisone therapy. *A.M.A. Arch. Dermat. & Syph.* 68:576, 1953.

fers with venous return. The condition usually affects the lower third of the leg. It occurs especially in young women and is characterized by edema and dusky redness.

ARTERIAL OBSTRUCTION

Conditions due to temporary or permanent vascular obstruction include diabetic gangrene, syphilitic arteriosclerosis with gangrene ischemic leg ulcers thrombo-angitis obliterans and senile arteriosclerosis with gangrene and decubitus ulcers.

Arteriosclerotic ulcers penetrate the deep fascia are usually of traumatic origin are often localized over the anterior and anterolateral surfaces of the lower leg and are painful. Secondary infection and a foul secretion are frequently observed. The clinical features of circulatory deficiency are apparent in the limb as a whole.

Prognosis depends upon the age of the patient, the duration of the symptoms and the extent of the involvement.

Diagnosis is made by lividity and coldness of the affected part, subnormal skin temperature and oscillometric readings.

Therapy Complete rest sympathicolytic drugs (Etamon, Priscoline) anticoagulants, measures to eliminate vasospasm abstinence from tobacco and local use of ulcer pastes (cod-liver oil and antibiotic) to meet individual requirements. Compression bandages must be avoided.

BED SORES

Bed sores (decubitus ulcers) are caused by prolonged pressure on dependent parts in those with lowered vitality poor circulation or spinal lesions. The condition usually occurs on the buttocks, over the trochanters, the heels and the elbows. Bed sores are common in debilitated patients with carcinoma, cord injuries, cardiac diseases, leprosy and trophic disorders who do not get sufficient nursing care to prevent the condition.

Prevention. Bed sores often can be prevented by the use of an air cushion or a water mattress to relieve the pressure frequent alcohol sponges the use of talcum powders, frequent changes in position and maintenance of nutrition and positive nitrogen balance (amino acids and high protein diet)

Treatment. In mild cases, scrupulous cleanliness and the use of borated talcum zinc-oxide ointment or compound tincture

of benzoin are usually effective. When necrosis occurs, the use of the infra red lamp and the application of epithelial stimulants or skin grafting are indicated.

VENOUS OBSTRUCTION

The common conditions associated with slowing of the venous blood flow are varicose dermatitis (gravitational, or hypostatic, eczema) and the varicose vein complex.

VARICOSE VEIN COMPLEX

This comprises a group of disorders of the lower extremities resulting from poor venous return stasis, phlebitis or their complications. It is associated with superficial or deep varicosities. The greater saphenous vein is incompetent in about 85 per cent of the cases as a result of defective valves. These may be destroyed partially by recurrent attacks of phlebitis or rarely are congenitally defective.*

For a valuable review of leg ulcers caused by defective venous return, arterial disease and ulcers associated with arthritis, see Anstog, S. T. *Leg Ulcers Their Causes and Treatment*, Boston, Little, 1955



FIG 167 Extensive bed sore with necrosis



FIG 164 Stasis eczema, with lymphedema and a subacute excretory dermatitis.

Clinical Description. The early symptoms of fatigue, edema and cramps, develop gradually. After a variable period the dilated veins make their appearance. If untreated they increase in number in size and become subject to complications.

STASIS DERMATITIS is a common end-result of the circulatory disturbance and usually makes its appearance on the lower third of the legs. The left extremity is involved more frequently. Sharply limited at first the dermatitis may spread upward to the knee or downward to the ankle or it may encircle the entire leg. Depending upon the duration and local factors, acute, subacute, or chronic stages may be present. Itching may be absent or it may be severe. Irreversible changes, including infiltration, ulceration, pigmentation and atrophy eventually take place. Varicose eczema often overlies a pool of blood, deep varicosities or thromboses, as shown by varicography.

Etiology. Stasis dermatitis, the basic cause of which is faulty venous return, may be initiated by trauma, thrombophlebitis, contactants, focal infection, cardiac weakness and postural defects.

The treatment of varicose eczema is outlined on page 82.

VARICOSE ULCERS are round or irregular ulcers, surrounded by infiltrated edematous or pigmented tissue. The lower third of the leg above the internal malleolus is a common site. Usually

there is a history of a chronic varicose dermatitis. Moderate pain is present in practically all cases. There is no tendency to spontaneous healing. The ulcers are single or multiple, shallow or deep reddish to purplish in color the floor is covered with a grayish exudate, and the edges are edematous and overhanging.

Complications due to stasis, anoxemia and the lowered resistance of the skin are common. Phlebitis is a complication in about 50 per cent of the cases. The infection may spread to the lymphatics and cause lymphedema which interferes with healing and conceals the varicosities. Periphlebitis purpura, subacute cellulitis and the irreversible sequelae of pigmentation, atrophy and scarring are seen frequently as complications.

Etiology Varicose veins are due to an inherent weakness of the vessel walls, poor muscular tone absence of valves below the sapheno-femoral junction thrombophlebitis, entropsois or flat feet. In women the condition often follows pregnancy as a result of mechanical back-pressure. Varicose veins often are associated with pelvic tumors and obesity. The condition is about equally prevalent in both sexes the average age is 50.

Diagnosis of varicose vein complex is made by the history of chronicity the presence of varicose veins, the typical localization and the condition of the surrounding tissues.

Differential diagnosis is to be made from traumatic and arteriosclerotic ulcers, ulcers of sickle-cell anemia pyoderma, late syphilis and tuberculosis. In erythema induratum the ulcers are usually multiple and affect the calf of the leg. They occur in younger individuals and begin as subcutaneous nodules which break down.

Treatment. Bed rest is important in all painful and edematous cases. Healing does not occur until the surrounding venous stasis, the underlying varicosities and the lymphedema are controlled. Flat feet and overweight should be corrected where possible, and any cardiac defects should be treated with appropriate remedies.

Infected cases may respond to wet dressings of benzalkonium chloride 1:5,000 Tyrothricin or hot boric acid.

Moderate stimulation of varicose ulcers may follow ultraviolet light therapy the use of a 2 per cent aqueous solution of gentian violet an irradiated cod-liver oil ointment, absorbable gelatin sponge powder or zinc peroxide. When edema or dermatitis is present surgical treatment should be withheld and elastic band-

ages should be used, encircling the leg from the toes to just below the knee in overlapping spirals. If local therapy is ineffective the ulcer may be covered with a rubber sponge and the rest of the leg may be supported by an elastic bandage. Large ulcerations may require skin grafts. Recurrent cases are treated by ligation of the great saphenous vein and its uppermost tributaries.

SUPPORTING THERAPY The keystone in the treatment of the varicose-vein complex is the removal of the varicosities which are the underlying cause.

Temporary measures consist of the use of varnishlike preparations (gelatin-zinc U.S.P. or the Unna boot) the general formula of which is as follows.

Gelatin	15
Glycerin	10
Zinc oxide	25
Water	50

Bandages containing this preparation (Gelocast or Varivane) may be used which, after application, adapt themselves to the limb before drying and hardening. They are applied with the lower limbs elevated and the patient at rest. Elastic adhesive bandages are also useful in controlling post thrombophlebitic edema. These should be removed once or twice weekly increasing the interval as the condition of the tissues improves.

SCLEROTHERAPY is a palliative method of treating varicosities but, unless skillfully done, may produce ulcerations in the skin and deep thrombi. Sclerosing solutions produce an irritative endovenitis, with the production of organized thrombi which contract later transforming the vein into a fibrous cord. Because of the presence of intercommunicating veins, this form of therapy does not produce permanent results. Synasol, Monolate and 5 per cent sodium morrhuate solutions are about equally effective. It should be noted that drug reactions may follow the use of these solutions in patients with an idiosyncrasy to them. The injections are made at weekly intervals until all the veins in the legs have been treated. After each injection a drop of flexible collodion is applied to the site to prevent leakage.

SURGICAL MEASURES. High saphenous-vein ligation at the level of the forna ovalis, and transection and ligation of all contributing veins to retard chronic passive congestion is indicated in all cases where faulty venous return is a factor.

Excision of the ulcer followed by split thickness grafts or pinch grafts is indicated in indolent ulcer recurrences following vein ligations.

Amputation is advisable in those cases of indurated leg with chronic indolent ulcers and tissues of lowered vitality which do not respond to vein ligation or skin grafts.

TREATMENT OF VARICOSE DERMATITIS (See p 83)

LIVIDO RETICULARIS

Livido reticularis is a vascular disturbance due to various causes, usually affecting the legs. It is characterized by a mottled bluish red network of dilated deep vessels.

The noninflammatory type is known as cutis marmorata. It is a functional vasomotor condition and occurs in normal individuals especially in children as a spasm of the arterioles resulting from exposure to cold. The hyperemia disappears temporarily when stroked to reappear slowly as arterial blood flows into the area.

The inflammatory types are usually persistent and secondary to syphilis and tuberculosis. Other forms may be associated with arteriosclerosis, periarteritis nodosa, anomalies of the blood vessels, endocrine and cardiac disturbances.

Treatment is that of the underlying cause.

Erythema ab igne is a related reticulated pigmentary condition produced by prolonged exposure to the heat of a fireplace, a radiator a hot water bottle or an electric pad. The meshwork lesions, which are reddish to brownish in color are limited to those parts exposed to the heat. Those with malnutrition and poor circulation are predisposed.

Treatment is unsatisfactory

TELANGECTASIA

This vascular disorder consists of tiny capillaries in normal or pathologic skin and may be caused by various external and internal factors.

The following types occur

Those on the face and neck in outdoor workers (chronic exposure to wind and sun)

Lesions on the chest in chronic emphysema and chronic chest diseases.

Pulsating lesions on the abdomen in chronic cirrhosis of the liver

Lesions on the face, mucous membranes, palms and fingers associated with recurrent bleeding and a hereditary history (familial hemorrhagic telangiectasia)

Lesions part of the clinical picture of certain skin disorders (rosacea, chronic lupus erythematosus, adenoma sebaceum, old scars and keloids, radiodermatitis, xeroderma pigmentosum, calcinosis cutis)

Lesions patchy in character consisting of dilated capillaries (portwine nevus, poikiloderma)

Lesions in normal individuals who flush easily (limited to nasolabial furrows)

These are dilatations of pre-existing vessels, without new vessel formation. The congenital types include Osler Weber Goldstein disease. The acquired types consist of cases associated with such diseases as lupus erythematosus and roentgen dermatitis cases due to exposure to wind and cold the traumatic nevus araneus (spider nevus) varices or ectasia composed of tufts of capillaries on the trunk or the extremities.

ERYTHEMA PERNIO

(See Chilblain p 277)

NURSING ASPECTS

Those diseases in which arterial obstruction is a factor require special observation and therapy. Patients with thrombo-angitis obliterans and arteriosclerotic gangrene are hospitalized in order to study the extent of the process and to prepare the case for surgery or special measures. The nurse should be familiar with prothrombin tests, the administration of Priscolline, heparin and Dicumarol.

Varicose-vein Complex. The nurse will be called upon to assist the physician in testing the extremity to determine the site of the venous insufficiency. The correct method of applying an elastic bandage must be mastered. Ointments applied to the legs should be covered with clean linen and a white cotton stocking or tubular gauze should be slipped over the extremity to keep it in place. Adhesive plaster must never be applied directly to the skin. Following a high saphenous ligation for varicose veins, the patient usually is advised to wear an elastic stocking, an elasto-plast adhesive bandage or a zinc-gelatin fixed dressing.

Bedsore in the aged chronically ill and undernourished may be prevented by the following measures use of oils rather than alcohol for massage keep sheets clean and free of wrinkles and foreign material frequent change of position use of air cushions applications of zinc stearate powder over pressure areas frequent observation of entire body for early signs of pressure sores and avoidance of trauma when using bedpans.

Pigmentations

THE MELANOCYTES	CHLOASMA
BLOOD-PIGMENT DYSCHROMIAS	EPHELIDES
PIGMENTATION FROM ABSORPTION OF DYES	ADDISON'S DISEASE
XANTHOSES	ACANTHOSES NIGRICANS
PIGMENTATION DUE TO HEAVY METALS	BERLOQUE DERMATITIS
ARSENIC	URTICARIA PIGMENTOSA
GOLD SALTS	DEPIGMENTATION (ACHROMIAS) OF THE SKIN
MERCURY	ALBINISM
BISMUTH	LEUKODERMA
SILVER	POSTINFLAMMATORY ACHROMIA
PIGMENTATION DUE TO FOREIGN SUBSTANCES	NURSING ASPECTS

The color of the normal skin depends upon its vascularity and thickness the amount of subcutaneous fat and upon the amount of pigment (melanin) in the basal-cell layer

Origin of Melanin. The normal pigment of the skin is melanin, a complex iron-free substance. Three basic substances are necessary for the formation of melanin (1) the oxidase tyrosinase a copper-protein complex attached to ultramicroscopic particles in the cytoplasm of the melanoblast, (2) a suitable substrate tyrosin or dopa and (3) molecular oxygen. Certain reducing agents, e.g., ascorbic acid and the sulfhydryls, probably hold the oxidases in check. Stimuli of various types (ultraviolet light, estrogens, the melanocyte-stimulating hormone of the pituitary gland 8-methoxypsoralen) can activate these reducing substances so that the melanin-forming oxidases can function.

Diagnosis. Special stains and histochemical and darkfield examinations are employed in cases where the type of pigmentation is undetermined.

THE MELANOSSES

The melanotic pigmentations are common conditions and vary in their importance from that of a freckle to that of Addison's disease. Any shade of yellow brown or black may be represented. Some of the melanoses are temporary conditions others are permanent depending upon the cause.*

Types. THE PRIMARY TYPES of melanosis (no previous inflammation) are usually internally in origin

THE SECONDARY TYPES result from various inflammatory diseases of the skin.

Pigmentation due to melanin may be localized or generalized. The localized forms usually are produced by chemical physical or inflammatory irritation the generalized varieties by any condition which stimulates the chromaffin system, thus increasing the amount of oxidizing enzyme.

THE GENERALIZED TYPES which are characterized by a diffuse pigmentation of the skin occur in visceral malignancy pellagra, malaria diabetes, syphilis, leprosy tuberculosis, hyperthyroidism hepatic disease Banti's disease, arsenical intoxication and melanocarcinoma.

THE LOCALIZED TYPES, which vary in size from a millimeter to several centimeters, occur in a large number of conditions which may be listed as follows

Physiologic Chloasma linea nigra and hyperpigmentation of nipples and genitalia in pregnancy

Racial Pigmentation of gums, palate and buccal mucosa in Negroes and persons of other dark skinned races.

Congenital Pigmented nevi, freckles, blue nevus, Mongolian spot neurofibromatosis incontinentia pigmenti, and xeroderma pigmentosum.

Physical Tanning or freckles from ultraviolet light, sunlight or x-ray therapy friction or heat (erythema ab igne)

Chemical Occupational dermatitis, with pigmentation from tar and creosote (*Rick's melanosis*) Irritating applications, including tincture of iodine mustard plaster and acids berlocque dermatitis from the action of the sun on oil of bergamot in perfumes and toilet waters and lime juice metallic absorption of bismuth or mercury in cosmetics.

*For an excellent discussion of the melanoses, see Becker S. W. Pigmentary diseases of the skin, Clinics 3:826-997 1944

Toxic Drug eruptions, especially those caused by arsenic, Mesantoin, thioracil ACTH and phenolphthalein.

Inflammatory The postinflammatory melanoses comprise a large group of pigmentary disturbances which are produced by vascular congestion pruritus or specific inflammations including lichen planus, dermatitis herpetiformis, gummas, cutaneous tuberculosis, pediculosis corporis (vagabond's disease) insect bites, measles and other exanthemata flat juvenile warts, pigmented basal-cell carcinoma and malignant acanthosis nigricans.

BLOOD-PIGMENT DYSCHROMIAS

This group includes disorders caused by venous stasis, injuries to the blood-vessel walls or direct toxic destruction of the erythrocytes. Purpura, dermatitis hypostatica Schamberg's disease, ochronosis, hemochromatosis and the pigmentation of pernicious anemia belong to this group. The discoloration is caused by an iron-containing pigment, hemosiderin.

PIGMENTATION FROM ABSORPTION OF DYES

The skin may become pigmented from the absorption of picric acid and picrates if they are used over a large surface or over a long period of time. The skin and the mucous membranes also may develop a yellow tint from the continued use of atabrine and acriflavine.

XANTHOXIS

This is a yellowish-orange pigmentation of the creases of the palms and the soles, the forehead, the rims of the nostrils and the postaural creases, resulting from the absorption of carotene (provitamin A) a vegetable pigment found in egg yolk, squash, carrots and oranges. The condition frequently occurs in diabetes, hypothyroidism and liver diseases from interference with carotene conversion into vitamin A and in those on restricted vegetable and fruit diets. After dietary correction the pigmentation disappears gradually. The diagnosis can be verified by finding a high level of carotene in the blood. The blood serum is golden yellow.

Differential diagnosis from jaundice is by absence of itching and by clear sclerae.

Treatment consists of removal of the cause.

PIGMENTATION DUE TO HEAVY METALS

Most of the generalized melanoses observed in the prepenicillin era were caused by the heavy metals used in syphilotherapy. Per

sonal idiosyncrasy and light sensitization are predisposing factors. Negroes especially tend toward heavy metal pigmentation.

Detection of Heavy Metals. The qualitative and the quantitative analyses of tissues for gold silver mercury bismuth and arsenic is an expensive and time-consuming affair. In many cases the results of the examination may be entirely negative in spite of a positive history and clinical findings.

MICROCHEMICAL EXAMINATION of stained tissue in cases of arsenical pigmentation, dermatitis and keratoses often fails to reveal the arsenic crystals even in typical cases.

THE DARKFIELD METHOD may be used to demonstrate the granules of silver around the glandular ducts and the blood vessels in tissue sections.

SPECTROSCOPY In medicolegal cases, spectroscopy of the incinerated tissues and direct spectroscopic examination of blood hair and nails may be necessary to establish the diagnosis.

ARSENIC

Inorganic arsenic may cause a diffuse or mottled ("rain-drop") pigmentation with or without hyperkeratoses of the palms and the soles. Fowler's solution is a common cause of this variety. The entire cutaneous surface in these patients should be examined at least once a year and any suspicious keratoses or nodules removed surgically. The clinical diagnosis should be verified by microscopic study.

Treatment is unsatisfactory.

GOLD SALTS

Gold salts, formerly used in the treatment of lupus erythematosus and various forms of tuberculosis and radioactive isotopes used in metastatic cancer may produce a diffuse slate-colored pigmentation (chryso-cyanosis). If the vein is missed and if a paravenous injection results the affected area becomes discolored by a bluish-green stain (chryxoderma).

MERCURY

Mercury when applied locally to the skin as an ingredient of cosmetics, may produce a slate-colored follicular pigmentation of the folds of the face and the neck in sensitive individuals.

BISMUTH

Generalized pigmentation from intramuscular injections of bismuth is rare but may follow exfoliative dermatitis resulting from its use. The mucous membranes are more apt to be involved, especially the gums. In these cases, gingivitis and stomatitis are prominent features. If the patient is receiving arsenicals also it may be difficult to make a diagnosis without a spectroscopic examination of the skin biopsy.

Treatment is unsatisfactory.

SILVER

Silver when it is deposited in the tissues, produces a peculiar slate-colored pigmentation known as argyria. Since the attention of the medical profession has been called to the danger of long-continued use of silver preparations, this form of pigmentation is now relatively uncommon. Argyria occurs in generalized and localized forms.

Varieties. THE LOCAL VARIETIES result from (1) accidental tattooing with silver salts and (2) the local use of Argyrol and silver-protein salts in diseases of the eye the nose the mouth and the throat.

THE GENERAL VARIETIES may follow (1) ingestion of the drug for relief of gastric and mental diseases, (2) absorption from amalgam fillings and sutures and (3) intravenous injection of radioactive silver isotopes. The latent period may be several months.

Differential Diagnosis. Argyria must be differentiated from cardiac and pulmonary cyanosis Addison's disease, bismuth pigmentation, polycythemia vera and methemoglobinemia.

Diagnosis is made by the history and the color of the pigmentation, which is blue on the exposed surface and ashen-gray on the unexposed areas. The nail beds also share in the pigmentation.

Prognosis is poor. The discoloration is a permanent condition.

Treatment is generally unsuccessful.

PIGMENTATION DUE TO FOREIGN SUBSTANCES

Tattooing may be decorative, accidental or occupational. In the decorative types, carmine and mercury salts are used. Occupational tattooing occurs in coal miners, grinders and workers in allied occupations. Accidental tattooing may result from the use of steel electrolysis or hypodermic needles, injuries or explosions

(carbon and gunpowder) In this type of pigmentation an attempt should be made to remove the foreign substance with a small sharp curet immediately after the accident.

Local applications of metallic salt solutions may be absorbed occasionally by injured tissues and cause pigmentation.

Treatment. Removal of tattoo marks is unsatisfactory. Small lesions may be removed by excision. Dermal abrasion therapy may be effective (see Acne Scars). The preferred treatment for the larger areas consists of scarifying or tattooing the outlines with 50 per cent tannic acid and then applying solid silver nitrate.



FIG 169 Tattoo in a Syrian woman. The patient was tattooed by her father at the age of 16 to prevent her from being kidnapped by unwelcome suitors.

CHLOASMA

Chloasma (liver spots, moth patches) is a common type of pigmentation usually affecting the face, especially the forehead and the cheeks. The discoloration varies from light yellow to dark brown and is more or less symmetrical.

Etiology The cause of chloasma of pregnancy is not known but Weiner lists the following theories: adrenal-cortical hypertrophy, vitamin C deficiency, increase in the estrogen levels, and a possible hereditary factor. Abdominal and utero-ovarian diseases produce chloasma through stimulation of the chromaffin



FIG. 170. Chloasma in a young woman with chronic pelvic infection.

system. Most of the cases occur in women in middle life, but some cases follow gynecologic operations. Hypervitaminosis A may produce a similar pigmentation. Exposure to sunlight accentuates the pigmentation. Frequently the cause cannot be determined.

Differential diagnosis is from pigmentation from cosmetics,

healed lupus erythematosus with minimal atrophy and early Addison's disease.

Treatment. Results are discouraging, although some cases clear up spontaneously after a variable period.

If the chloasma is not the result of pregnancy the patient should be examined for pelvic, endocrine and hepatic disease.

Benoquin ointment (Elder) a 20 per cent monobenayl ether of hydroquinone product may be useful in some cases. It is applied 2 or 3 times a day for 1 to 4 months unless sensitization occurs. Exposure to direct sunlight must be avoided and it must not be used near the eyes. Contact with the normal skin and mercurial ointments should be avoided to prevent undesirable bleaching effects.

EPHELIDES

Ephelides (freckles) are common small yellow or yellowish-brown pigmented macules. The lesions usually occur on the face and the back of the hands. They may be found occasionally on the covered areas, including the arms the trunk and the buttocks (cold freckles). Freckles may be scattered and hardly visible or they also may be grouped closely to constitute a cosmetic defect.

LENTIGO MALIGNA is a black or bluish-black nevoid freckle which if irritated, may develop into nevocarcinoma. It usually occurs in patients over 50 years of age. It is part of the picture of xeroderma pigmentosum and is larger than the common freckle.

LENTIGINES are congenital freckles that occur on the covered parts of the body and are noted soon after birth. They must be differentiated from urticaria pigmentosa and flat pigmented nevi.

Etiology The condition reaches its height during adolescence and then fades gradually. Blondes and red-haired individuals are predisposed. Although freckles disappear during the winter months, they may be demonstrated in the skin as permanent pigmentations with the aid of a Wood light. They are considered to be congenital nevi which are stimulated by the actinic rays. Acquired types may follow exposure to x rays or ultraviolet light.

Differential diagnosis from urticaria pigmentosa, xeroderma pigmentosa and early neurofibromatosis usually is not difficult.

Histology A freckle consists of a localized collection of fine melanin granules in the basal cells of a normal epidermis.

Treatment should be discouraged. Results are indifferent and often unsuccessful. Solutions or creams containing hydrogen peroxide have little effect on the lesions. Acids never should be used because of the danger of scars and malignant degeneration. Light freezing with solid carbon dioxide may succeed if the lesions are superficial. Dermal abrasive therapy may be successful if skillfully performed.

ADDISON'S DISEASE

Addison's disease is a syndrome due to deficient production of adrenocorticosteroids resulting in various symptoms including a diffuse negroid pigmentation of the skin and the mucous membranes. It is associated with low blood pressure, anorexia, weakness, loss of weight and vague abdominal complaints. Low blood chlorides and increased urea are constant findings.

Diagnosis. The clinical symptoms and the typical pigmentation are characteristics. X-ray of the adrenals may reveal calcification. Blood serum examination reveals low sodium and chloride levels and a rise in potassium. In borderline cases the water test, the 4-hour ACTH test and a therapeutic trial with desoxy corticosterone acetate are useful.

Differential diagnosis is from racial pigmentation, pellagra and general pigmentation from abdominal tuberculosis or malignancy.

Etiology The syndrome may be the result of the following conditions, affecting the adrenal cortex: tuberculosis, hemorrhage, atrophy, carcinoma, thrombosis, embolism or calcification.

Pathology Numerous chromatophores and melanin granules are found in the basal cells.

Treatment. Hydrocortisone therapy must be individualized with a starting dose of 150 to 200 mg. daily and gradually tapering off to a maintenance dose of 10 to 15 mg. If indicated 4 to 6 Gm. of sodium chloride and up to 3 mg. desoxycorticosterone (D.C.A.) is injected intramuscularly daily or implanted as a pellet. If a crisis appears, the dose of hydrocortisone is increased 100 to 200 mg. daily.

Large doses of ascorbic acid may reduce the pigmentation.

ACANTHOSIS NIGRICANS

Acanthosis nigricans is characterized by a rugose pigmentation of the axillae, flexures, neck, and the sub-mammary, the cubital

or the *genito-crural regions*. The onset may be *sudden or gradual*, the juvenile form usually appearing at puberty *

Clinical Description. The eruption consists of yellow black or brown streaks or patches, associated with a verrucous or papillomatous hypertrophy. In mild cases the eruption may be overlooked or its significance misinterpreted. Alopecia nail changes and an eruption of pigmented macules are often present in extensive cases. Most of the juvenile types have been reported in obese children.

Etiology The juvenile types are of endocrine or metabolic origin. Visceral malignancy and hepatic cirrhosis are factors in the adult type.

Pathology Marked acanthosis is present with a moderate or marked deposit of melanin in the basal-cell layers. The benign and the malignant types present similar pathologic changes.

Differential diagnosis is from pigmentation following irritants, Fox Fordyce disease linear nevus and Darier's disease.

Prognosis. The juvenile types tend to disappear spontaneously but should be observed for a protracted period. When the disease occurs in adults a complete physical examination followed by an exploratory laparotomy should be made to discover the malignancy

Treatment. Thyroid and large doses of vitamin A are indicated in the juvenile types after a thorough medical examination. Surgical consultation must be obtained in the adult cases in order to investigate the presence of visceral malignancy

BERLOCQUE DERMATITIS

Berlocque dermatitis is a rare type of streaked pigmentation following contact with insect sprays, perfumes or toilet waters containing essential oils. Copper impurities may provoke the reaction although personal idiosyncrasy is an important factor. Following exposure to the sun the areas to which the solutions were applied become pigmented. A long latent period may occur between the application of the perfume and the appearance of the discoloration.

Treatment. Benoquin ointment may reduce the pigmentation if used for several months. In 10 per cent of the cases, a local sensitization may interfere with its continued use

*For an excellent review of the subject, see Curth, H. O. Acanthosis nigricans and its association with cancer Arch. Dermat. & Syph. 57:1158, 1948.

URTICARIA PIGMENTOSA

Urticaria pigmentosa is a rare disease which is characterized by a more or less symmetrical generalized eruption of numerous pigmented macules or nodules.

Clinical Varieties. (1) Juvenile and (2) adult or acquired type.

THE JUVENILE TYPE usually begins in infancy as an evanescent eruption of bullae or inflammatory smooth, pink macules which are round or irregular in shape. In size, the lesions vary from that of a pea to that of a silver dollar. As the disorder progresses the lesions tend to become yellow or dark brown in color. In some cases the early lesions are raised above the surface,



FIG. 171. Urticaria pigmentosa (juvenile type) with spontaneous involution after 5 years.

giving a nodular character to the growth. When the condition is fully developed the trunk, the extremities and even the face may be involved. The human leopards of the circus sideshow are examples of urticaria pigmentosa.

THE ADULT TYPE appears at any time after puberty. The eruption does not differ from the juvenile form except that telangiectasia may be present, and urtication is usually absent.

Itching may or may not be present. If the lesions are stroked they may urticate a phenomenon from which the name of the

disease is derived. Urtication is usually present in the early cases and tends to disappear as the disease progresses. General enlargement of the lymph nodes and dermographism occur in about one half of the cases. Bone involvement is rare and may represent collections of mast cells.

Etiology The cause of the disease is unknown. Mast cells in the bone marrow and the lymph nodes point to a systemic factor.

Both sexes are equally affected. Exposure to the sun intensifies the pigmentation and may produce new lesions. About 50 per cent of the cases occur within the first six months of life.

Differential Diagnosis. The disease must be differentiated from postinflammatory melanosis, juvenile xanthoma, multiple pigmented nevi and phenolphthalein drug rashes.

Course and Prognosis. The disorder tends to disappear spontaneously after several years. There are three stages in the evolution of the condition (1) the stage of activity which lasts for about one year (2) the stage of inactivity which persists for from three to five years (3) the stage of retrogression, which is of several years' duration.

Pathology The characteristic feature of the disease is a marked infiltration of the corium, with or without scattered basophilic mast cells, which usually are found around the blood vessels in the upper part of the corium. Mast cells also may be found in the uninvolved skin and in the lymph nodes. Their function appears to be concerned with the production of heparin, an anticoagulant.

Treatment. No specific measures are available. Antihistaminics may control the pruritus. Toluidine blue, an heparin antagonist, has been used experimentally but results are inconclusive.

DEPIGMENTATION (ACHROMIAS) OF THE SKIN

Loss of pigment (achromia or hypochromia) may be congenital (albinism). It may follow inflammatory dermatoses (chronic lupus erythematosus, burns, radiodermatitis), atrophic dermatoses or it may be idiopathic in origin (vitiligo).

ALBINISM

Albinism is a congenital type of generalized leukoderma (see p. 463)

LEUKODERMA

Leukoderma is an acquired patchy or maplike loss of pigment in an otherwise normal skin following some antecedent inflammation. The hairs in the involved areas also share in the process. The depigmented areas are characterized by concave borders which in some cases seem to be lined by a threadlike inflammatory border. The condition is especially noticeable in dark skinned individuals. It is more evident during the summer months, when the normal skin is hyperpigmented in contrast with the depigmented areas.

Vitiligo is a functional idiopathic type of leukoderma: bilateral or symmetrical, insidious in onset, often appearing on the upper



FIG. 172 Vitiligo following "nervous breakdown"

eyelids, the genitals, the anterior wrists and the forearms, ankles and the neck. The symmetrical nature of the depigmentation in most cases suggests a neuropathic causation of central origin.

Etiology The cause of the disorder is unknown. For some undetermined reason the melanocytes are unable to convert tyrosine into melanin. Both sexes are equally affected. Negroes are apparently predisposed although they are more apt to seek treatment than are white patients. The disease may develop at any age. However in most cases, the pigment exhaustion appears at puberty. Some cases follow sunburn, mental shock or laparotomy.

The Cutaneous Lymphoblastomas (Lymphomatoid Diseases)

MYCOSIS FUNGOIDES
HODGKIN'S DISEASE

LEUKEMIA CUTIS
LYMPHOSARCOMA
NURSING ASPECTS

General. The malignant lymphoblastomas are rare neoplastic disturbances of the hematopoietic and the reticulo-endothelial systems. These tissues consist of (1) reticulum cells of the spleen and the lymph nodes (2) the reticulo-endothelial cells of the lymph sinuses of the lymph nodes and (3) the blood sinuses of the spleen the liver the bone marrow the adrenal cortex and the hypophysis.

The lymphoblastomas include mycosis fungoides, Hodgkin's disease leukemia cutis (myelogenous lymphatic, monocytic and their subleukemic forms) and lymphosarcoma, all of which are strikingly similar in their cutaneous reactions and involvement. They may be genetically related diseases and arise from a parent cell, the primitive reticulum cell, which is found in the upper corium in the leukemias and in the lymph nodes in Hodgkin's disease and lymphosarcoma.

Diagnosis of the lymphoblastoma group depends on the finding of specific changes in the skin the bone marrow or the lymph nodes, evidence of bone involvement, specific blood picture in the leukemias and clinical course.

Various mutations and transitions may occur from one disease to the other but the whole group has the following characteristics in common (1) fatal prognosis (2) difficulty of arriving at an accurate clinical diagnosis without biopsies, hemograms and sternal-puncture studies (3) temporary susceptibility to radiation and nuclear toxins (4) tendency to involve viscera and bones.

MYCOSIS FUNGOIDES

This lymphomatoid disease is a serious disorder of unknown etiology which is characterized by superficial inflammatory lesions in the beginning followed by tumorlike growths and a tendency

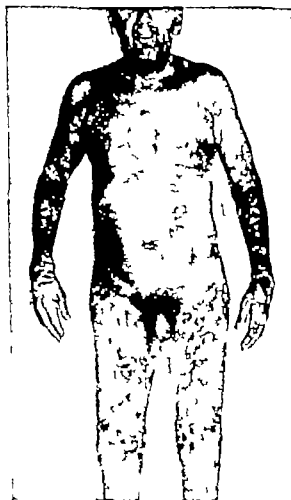


FIG. 173. Ulcerative stage in mycosis fungoides (Dr Vincent J. Ryan's case)

to visceral involvement. In some cases the early lesions may resemble psoriasis but there is no relation between the two diseases.

There are three stages of the disease. (1) In the premycotic (eczematoid) stage the eruption is toxic or nonspecific and may be transient or may persist for months or years. It is characterized by intense pruritus. Exfoliative dermatitis or lichenoid, psoriatic or parapsoriatic lesions and alopecia of the scalp may be present. Eczematoid or papular lesions of no set pattern may occur also.

(2) The infiltrative stage is characterized by succulent, rose-pink, yellowish or brownish plaques of various sizes, bizarre "horse shoe" shapes and raised borders. Central clearing is present in many of the older lesions. In this stage the disease is persistent and is refractory to any treatment but radiation.

(3) In the tumor stage the disease consists of firm, irregular mushroomlike growths which eventually break down and form horseshoe-shaped ulcers. Sepsis, visceral involvement and increasing weakness occur in the final stages.

Pathology In the early stages, the histology is that of a nonspecific inflammatory reaction. Later the changes consist of a polymorphous infiltrate around the blood vessels between the connective tissue bundles reaching up into the epidermis. The infiltrate consists of round cells, eosinophils, epithelioid cells and polymorphonuclear leukocytes. Clumping of the endothelial cells is a diagnostic feature. All the structures in the corium, with the exception of the blood vessels, are destroyed in the late stage. There is no characteristic blood picture.

Prognosis. Death occurs in from 6 months to 6 years, in spite of remissions from toxemia, cachexia or intercurrent disease. The younger the patient the worse the prognosis.

Treatment. Filtered x ray radiation overcomes the itching and melts the tumors in the early stages. However radiation may produce toxic symptoms from the breaking down of the tissue. Leukopenia may occur also since extensive radiation depresses the bone-marrow function and drives the leukocytes from the periphery to the splanchnic areas. Large doses of x-rays may eventually cause the tumors to become radioresistant. Blood transfusions are necessary in the stage of cachexia.

Antimony compounds (20 intravenous injections of the 1 per cent potassium tartrate 3 times weekly) triethylene melamine (5 mg every other day for 6 doses in courses 1 or 2 weeks apart)

fever therapy the nitrogen mustards and urethane also may give temporary benefit in roentgen-ray-refractory cases. ACTH and hydrocortisone may cause temporary involution of the tumors and control the pruritus. Chloroquine phosphate has been used with inconclusive results at this time.

HODGKIN'S DISEASE

Hodgkin's disease is a chronic generalized infection involving the lymphoid tissues, characterized by a progressive and painless enlargement of the lymph nodes, secondary anemia a moderate and irregular fever night sweats and weakness. The primary tumor may arise in the lymph nodes of the neck, the axillae, the inguinal area, the chest or the abdomen. Cutaneous manifestations occur in one third of the cases. These consist of (1) nonspecific skin lesions, including pruritus, papules, urticaria, lichenification, ichthyosiform scaling of the legs, scaling of the palms and the soles, brownish-gray pigmentation bullous lesions, alopecia and nail changes and (2) specific lesions including plum-colored nodules tumors or ulcers which are rare.

Pathology The cutis is infiltrated with lymphocytes, eosinophils, epithelioid and characteristic giant cells (Sternberg Reed). Splenomegaly is present in 50 per cent of all cases. Visceral lesions include foci in the lungs and the bones. The blood picture consists of a decrease in the lymphocytes but an increase in the monocytes. The lymph nodes are characterized by a destruction of the normal architecture a polymorphous infiltrate, consisting of eosinophilic leukocytes and Sternberg Reed cells, hyperplasia of lymphocytes swelling of the vascular endothelium, necrosis and fibrosis.

Etiology The cause is unknown. The disease is $2\frac{1}{2}$ times more common in males in the 20-year-old to 40-year-old age group.

Diagnosis is made by biopsies and imprints of the lymph nodes and the skin and by roentgenograms of the chest, the abdomen and the bones to determine the presence of visceral involvement. The blood is normal except for a progressive secondary anemia. Late in the course of the disease moderate eosinophilia and leukocytosis are present.

Course. The acute cases are rare and run a febrile course, with death occurring within 6 months. The chronic cases have fre-

quent remissions, with death occurring within 5 years. The benign cases may live for 25 years. Death usually occurs from pressure symptoms, inanition or lymphosarcomatous changes.

Treatment. X ray radiation produces a temporary reduction in the size of the nodes. General radiation may be given at the same time for its systemic effect. Fowler's solution may be prescribed between courses of x-ray treatment until improvement occurs. In some cases courses of nitrogen mustard (8 mg. I.V.,



FIG. 174. Gingivitis in acute leukemia (Dr. Louis J. Frank's case)

well diluted with normal saline) given every other day for 3 doses may give symptomatic relief.

Nitrogen mustards often produce short remissions but require frequent blood studies, as does prolonged x ray therapy. ACTH and hydrocortisone may produce temporary remissions in selected cases.* Transfusions are necessary as the disease progresses.

LEUKEMIA CUTIS

This type of lymphoblastoma is a disorder of the lymphoid or the myeloid tissues. It is characterized by (1) definite changes

* For a more complete review of therapy consult Black, M. M. and Speer, F. D. *Therapy of the lymphomas and allied diseases*, New York State J. Med. 52: 1668, 1952.

in the number and the character of the leukocytes with a preponderance of immature forms, (2) an increased basal metabolic rate, (3) anemia (4) tendency to spontaneous hemorrhage, (5) skin and visceral infiltrations, (6) frequent enlargement of the liver and the spleen and (7) generalized adenopathy. Clinically the skin manifestations consist of (1) toxic eruptions or (2) true

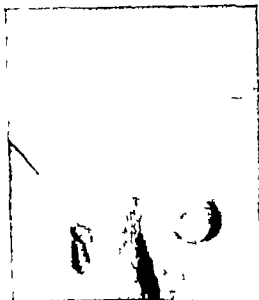


FIG. 173 Monocytic leukemia (nodular type) in 47 year-old woman. The white cell count varied from 23,000 to 47,000, and the monocytes, from 14 per cent to 28 per cent.

leukemic infiltrations, characterized by solitary or multiple reddish-brown scaly erythrodermic areas, infiltrated or edematous, brownish-red nodules or plum-colored tumors, ulcers or areas of necrosis.

Bluefarb† lists the following as early manifestations of this group: pallor, prurigo, pigmentation, pemphigoid lesions, purpura, pityriasis rubra, postganglionitis (herpes zoster), phlebitis, pru-

†Bluefarb, S. M. Nonspecific cutaneous manifestations of the leukemia lymphoma group and internal carcinoma. Postgrad. Med. 16:16, 1954.

ritus, poikiloderma and pyoderma. To this group may be added seborrhealike eruptions.

Visceral involvement takes place in the bones, the mouth and the pharynx, the eyes and the central nervous system. Diffuse lymphadenomatous erythroderma, a symmetrical generalized infiltrated pruritic exfoliative dermatitis is a rare complication in chronic types.

Etiology The disease the cause of which is uncertain has both chronic inflammatory and neoplastic characteristics. Suggestive factors are (1) virus infection (2) long-continued irritation of the reticulo-endothelial system (3) continuous exposure to radiation and (4) genetic factors *

Pathology The fundamental changes are in the tissues not in the blood. The infiltrate which may be diffuse, periglandular or perivascular should be studied carefully. The lymph nodes show a disturbance of their normal architecture.

Types There are three types (1) Lymphadenosis, or lymphatic leukemia this type is associated with the cutaneous manifestations previously described. The skin manifestations usually precede the characteristic blood picture. (2) Myelosis, or myelogenous leukemia. In this type skin lesions are rare. (3) Monocytic leukemia this type is characterized by pruritus, erythemas, papules and persistent grayish or purplish nodules, ulcers and ecchymoses.

Diagnosis. In an elderly patient, a generalized erythroderma associated with intense pruritus and adenopathy should be considered lymphoblastomatous until proved otherwise. Blood and bone-marrow studies by an expert hematologist are necessary to determine the exact type. An accurate diagnosis is often impossible until postmortem. Repeated hemograms and biopsies are useful and permit possible mutations to be discovered. Cutaneous types of the disease often have subleukemic phases (normal white count or leukopenia) showing qualitative but no quantitative changes, because the fundamental lesion is found first in the lymphoid tissue of the skin or the lymph nodes.

The peroxidase test is a useful laboratory procedure to aid in the differentiation of the myelogenous type from the lymphatic form.

*For a more detailed study of the problem see Furth J. Recent studies on the etiology and nature of leukemia, *Blood* 6:964 1951.

Prognosis should be guarded, since it is doubtful whether any type of therapy actually can prolong life. Death usually occurs within five years. Sturgis* emphasizes the following clinical features of the leukemias as influencing the prognosis: the impairment of the various tissues as a result of the characteristic leukemic infiltration; the gradual anemia; lessened resistance to infection; a hemorrhagic tendency as a result of destruction of the megakaryocytes in the bone marrow with a resulting decrease in the circulating blood platelets.

Treatment. X-ray therapy is the treatment of choice. Total body irradiation is necessary in the extensive cases with special attention to the long bones, the spleen and the lymph nodes. The dosage should be kept as low as possible, the objective being the maintenance of freedom from symptoms rather than a reduction in the white-cell count. Blood transfusions are necessary in the late stages to combat the anemia. Large doses of vitamins A and D are useful when itching is present. Radioactive phosphorus, folic acid antagonists (aminopterin in 2-mg. daily doses for adults for a few days if no bleeding or leukopenia results), nitrogen mustards and triethylene melamine (1 to 5 mg. daily for a total dosage of 12 to 25 mg. if no side effects occur) are useful in the chronic types but toleration, clinical response and length of remission vary in the individual case. ACTH and hydrocortisone may produce temporary remissions in selected cases. Antibiotic therapy is important to control infection which often is a serious complication.

LYMPHOSARCOMA

Lymphosarcoma is a true primary neoplasm of the lymphoid tissues. In some cases there may be a leukemic phase.

Types. (1) Giant follicular lymphoma, (2) malignant lymphocytoma and (3) the reticulum cell.

Clinically lymphosarcoma may resemble Hodgkin's disease. The onset consists of a gradual painless enlargement of the affected glands. The cervical, the axillary, the mediastinal and the abdominal glands usually are involved. Retroperitoneal and mediastinal metastases are common. Skin metastases are rare and consist of papules or purplish deep subcutaneous nodules.

*Sturgis, C. C. Some aspects of the leukemia problem, J.A.M.A. 130: 1331-1336, 1951.

Diagnosis is made by repeated biopsies and blood studies to eliminate leukemia or Hodgkin's disease. Herpes zoster due to glandular pressure may be an early sign.

Pathology Masses of abnormal lymphocytes are present in the nodes. The lymph-node architecture is destroyed. Mitotic figures are prominent and numerous.

Prognosis is poor. Death occurs within five years from cachexia, pressure symptoms, secondary anemia or intercurrent infection.

Treatment. Radical excision, followed by prophylactic x-ray radiation, is the method of choice. Destruction of the spleen by deep x-ray therapy may retard the progress of the anemia, which is progressive and is not benefited by transfusion. Nitrogen mustards and ethyl carbamate (urethane) may result in definite clinical improvement, but results are variable and unpredictable.

NURSING ASPECTS

These patients are hospitalized for diagnostic studies and to receive special therapy. General nursing care, permanganate baths if fetid secretions are present, a high-vitamin high-caloric diet, to maintain nutrition, cradles to avoid pressure of the bed-clothes and the avoidance of chilling and infections are important. The nurse should be familiar with the techniques of administering nitrogen mustards, radioactive phosphorus, triethylene melamine and blood transfusions. The nuclear toxic drugs must be used with caution since serious reactions may occur. Frequent observation and laboratory controls are necessary to avoid complications.

Dermatitis Exfoliativa

PRIMARY TYPES

CONGENITAL ICTHYOSIFORM

ERYTHRODERMIA

INFANTILE ERYTHRODERMIA

CHRONIC PARAPSORIASIS

SECONDARY TYPES

SECONDARY EXFOLIATIVE

DERMATITIS

MALIGNANT ERYTHRODERMIA

NURSING ASPECTS

DERMATITIS exfoliativa is a term used to designate a group of rare eruptions of variable etiology characterized by (1) universal or generalized involvement of the skin, (2) inflammatory erythema of variable degree (3) persistent scaling and (4) pruritus of variable intensity. In some cases the erythema is more prominent in others the scaling. They are divided into two groups. (1) The primary types include congenital ichthyosiform erythrodermia, parapsoriasis, scarlatiniform erythema, dermatitis exfoliativa neonatorum (Ritter). (2) The secondary types include reactions to such drugs as the arsenicals, gold bismuth etc. reactions following certain dermatoses, such as lichen planus, psoriasis, eczema, pityriasis rubra pilaris, etc., and the erythrodermias associated with the lymphoblastomas. About 25 per cent of all secondary types ultimately become lymphoblastomas. Malignant erythrodermia is the generalized or universal, erythrodermatous phase of the lymphoblastomas in which the clinical picture is not diagnostic and in which insufficient scaling to justify the term "exfoliative dermatitis" is present.*

PRIMARY TYPES

CONGENITAL ICTHYOSIFORM ERYTHRODERMIA

This rare type of erythrodermia is similar to ichthyosis but with erythema. Involvement of the scalp and the flexor surfaces and large plaque formation. Two forms are recognized (1) the

*The pathologic aspect of the various types is discussed by Montgomery II. Exfoliative dermatitis and malignant erythrodermia, Arch. Dermat. & Syph. 27:253, 1933.



FIG. 176. Ritter's disease, a rare type of bullous impetigo with extensive exfoliation. (Dr. Leon Goldman's case)

dry with a "varnished" appearance, and (2) the moist, which is associated with bullous formation.

Etiology The disease is a congenital dysfunction of the epidermis with a possible endocrine relationship. Most cases are familial; some are hereditary.

Pathology Section shows marked hyperkeratosis, areas of parakeratosis, a hypertrophic granular layer, acanthosis and an inflammatory infiltrate in the corium.

Diagnosis is made by a history of duration since birth, characteristic pathology and localization of the lesions.

Differential Diagnosis. Ichthyosis does not involve the flexures, and the characteristic redness is absent. Juvenile pityriasis rubra pilaris is primarily follicular.

Prognosis is poor.

Treatment is similar to that of ichthyosis. In some cases, vitamin A (100,000 units daily) for several months, if tolerated, may be beneficial.

INFANTILE ERYTHRODERMIA

Dermatitis exfoliativa neonatorum (Ritter) is characterized by a generalized erythema and scaling with bullae and crusting. Probably it is a variant of bullous impetigo. It occurs frequently in children in foundling institutions. Recovery usually occurs in three weeks unless there are septic complications. Bacitracin or neomycin ointments are useful.

SCARLATINEFORM ERYTHEMA

(See p 38)

CHRONIC PARAPHOSIASIS

This is a rare condition which includes a group of lichenoid, maculopapular and psoriasiform resistant dermatoses of unknown etiology. In spite of the name they have no connection with psoriasis. Clinically they sometimes are confused with the lymphoblastomas. Pruritus is absent in all types.

1 Lichenoid Type. The chronic type bears a faint resemblance to lichen planus, but itching is absent. The mucous membranes are never involved. The lesions are papular bluish-red or brownish-red and usually are found on the trunk and the lower extremities.

2 Guttae or Maculopapular Type. These lesions resemble secondary syphilis. They are pink or brownish-red in color and are covered with a fine scale.

3 Plaque Type. These are round, irregular or reticular orange-red pea-sized to saucer-sized psoriasiform patches. The lesions persist without change for years itching is absent infiltration is slight and response to treatment is poor. The scalp and the nails are not affected the scaling is not silvery scraping does not elicit capillary bleeding and the distribution is not that of psoriasis.

Pathology The pathologic picture is not specific.* Parakeratosis is minimal there is slight exocytosis in the prickle-cell layer papillomatosis is absent and there is a moderate infiltration close to the basal-cell layer.

Prognosis is guarded. Few cases clear up spontaneously.

Treatment is similar to that used in psoriasis. The chronic types may respond to vitamin D₂ (calciferol) 50,000 units t.i.d. but hypervitaminosis too must be avoided.

SECONDARY TYPES

SECONDARY EXFOLIATIVE DERMATITIS

Secondary exfoliative dermatitis, which is a disturbance of the keratinization cycle, is characterized by a generalized hyperemia which begins on the flexures and then becomes generalized, de-

Lever W F Histopathology of the Skin, ed. 2, p. 104 Philadelphia, Lippincott, 1954



FIG. 177 Acute exfoliative dermatitis following the fourth injection of neoursphenamine.

veloping into an inflammatory infiltration. After a variable length of time, exfoliation occurs. The scales are thin and grayish with the underlying skin smooth and red. The face, the legs and the ankles are apt to be edematous. There is a generalized adenopathy. In most cases there is marked pruritus, with hyperesthesia of the skin. Chilliness and loss of strength are marked complaints. The hair and the nails may be shed several weeks after the onset of the disease. Abscesses and furuncles, conjunctivitis, rhinitis, stomatitis, bronchitis and colitis from involvement of the mucous membranes may complicate the picture. A marked depression is usually present. Remissions are common.

Etiology. This type is a cutaneous reaction to various noxae in a susceptible individual. The eruption occurs secondarily to

applications of strong irritants, e.g. tar chrysarobin, or ammoniated mercury or it may develop following the internal administration of penicillin, heavy metals gold, antipyrine, sulfonamides antitoxins etc. In some cases the disease is secondary to such dermatoses as psoriasis, seborrheic eczema, disseminated neurodermatitis and contact dermatitis, which may react to incorrect or too-vigorous treatment.*

Pathology Frequent biopsies are necessary to rule out lymphoblastomas. The pathologic picture of secondary exfoliative dermatitis depends upon the nature of the previous dermatosis. Erythema psoriaticum is similar to psoriasis but is characterized by more intercellular edema. Erythema seborrheicum differs from psoriasis in that definite areas of spongiosis are present. Exfoliative dermatitis from lichen planus parapsoriasis, etc., contains features of the characteristic pathology of the original disease. The atypical cases resemble the pathology of eczema, with hyperkeratosis, spongiosis of the rete and pyknosis. In the eczema cases there is a spotted parakeratosis. In psoriasis, it is more uniform. Mitotic figures, which are observed frequently in psoriasis, are rare in eczema. On the other hand spongiosis, which is a characteristic feature of atopic eczema, is usually absent in psoriasis.

Diagnosis. The history should be studied to determine the etiology. Chemical studies should be made on the hair, the nails, the scales and the blood for the presence of arsenic, lead, bismuth, gold, nickel etc. Biopsy is important and is helpful in ruling out the lymphoblastoma group.

Prognosis. With the best of medical care, cure requires from 2 months to a year. Complications include nephritis, bronchopneumonia and secondary anemia.

Treatment. ACTH often controls acute episodes. After several days it should be discontinued, and hydrocortisone should be substituted to maintain improvement. If heavy metals are found by chemical studies, ACTH or cortisone combined with BAL may be given. Powder baths or colloidal baths are helpful as are such preparations as zinc cream boric acid ointment, calamine cream or equal parts of lime water and olive oil. A salt free diet should be prescribed. The urine should be examined at frequent intervals for evidence of kidney irritation. Hospitalization and careful nursing

*For an excellent review of the subject, see Wilson, H. T. H., Exfoliative dermatitis, *A.M. A. Arch. Dermat. & Syph.* 69:577 1954.

are important requisites for recovery. In cases that do not respond to the above measures, a course of intravenous triple typhoid vaccine injections (*see* p. 605) may be effective.

Protein hydrolysate therapy for several weeks is important in order to supply amino acids to maintain nitrogen balance, to prevent malnutrition and hypoproteïnemia, to protect the liver and to promote normal skin metabolism. Multiple vitamin therapy is important also.

MALIGNANT ERYTHRODERMIA

The majority of the cases of pityriasis rubra (Hebra type) are lymphoblastomatous in origin.

Clinical Features. The erythrosquamous eruption begins first in the flexures and after months or years becomes universal. The skin complications include abscesses, profuse scaling leaving a smooth cyanotic skin pigmentation ulcerations over pressure points and atrophy over the joints. In the later stages, the skin becomes taut, and ectropion or fissures usually develop. Marked pruritus and chilliness are characteristic symptoms. There is shedding of the nails and alopecia. Death may occur from emaciation, persistent diarrhea, bronchopneumonia, pulmonary edema, nephritis or exhaustion.

Etiology. The condition is a manifestation of the lymphoblastoma group.

Differential diagnosis is made by repeated blood, bone marrow, lymph node and skin studies. A uniform dense infiltrate, consisting mostly of lymphocytes is diagnostic of leukemia cutis. Mycosis fungoides is characterized by an infiltrate, composed of a multiplicity of cell types. The infiltrate consists of eosinophils, polymorphonuclear leukocytes and connective-tissue cells of the vessel walls. Clumping and pyknosis are characteristic features. In Hodgkin's disease a polymorphous infiltrate without polymorphism of the individual cells, and the presence of Dorothy Reed or Sternberg giant cells are of diagnostic value. Eosinophils in the tissue or the blood is of no diagnostic importance since it occurs in several benign dermatoses.

Diagnosis is based on the following findings: (1) rapid extension, (2) general or universal involvement, including palms and soles, (3) vivid redness, (4) persistent exfoliation, (5) branny laminated or flaky scales, (6) atrophy over joints, (7)

variable itching and chilliness, (8) constitutional involvement and (9) resistance to treatment.

Prognosis. As stated above, the outcome depends upon the type of the disease. The lymphoblastomatous varieties are often fatal. Internal complications and albuminuria are serious omens. The prognosis in all cases should be guarded because of the possibility of relapse.

Treatment. Patients with chronic exfoliative dermatitis require a high-caloric diet vitamin A and tonic measures to support the nutrition. Calamine liniment or boric ointment should be used to keep the skin soft. Oatmeal baths or powder baths are soothing to most patients. X-ray therapy reduces the pruritus and prevents proliferative changes in the flexural creases. ACTH and hydrocortisone should be given a thorough trial in all cases. If there is no response from the standard dosage, the dose should be doubled if there are no contraindications. Some cases, unfortunately do not respond.

Resistant cases may respond to fever therapy (triple typhoid vaccine IV in doses of 10 to 120 million every other day) or to a change to a warm climate.

NURSING ASPECTS

Patients with acute and chronic erythrodermas should be hospitalized, since proper nursing care is important for their comfort and recovery. These diseases are characterized by a general redness of the skin extreme scaliness, severe itching as a rule, irritability chilliness and various systemic symptoms. Even though the room is warm, sufficient covers and hot water bags should be supplied to keep the patient comfortable. The hair and the nails should be kept short. The nurse should wear gloves when giving the powder the colloid or the oil baths, the type depending upon the stage of the disease. A nutritious diet, hot drinks to encourage sweating, removal of shedding epidermis from the palms and the soles, routine care of the eyes, the mouth and the nose to avoid infection daily change of bed linen and precautions to avoid "catching cold" are important. During the acute stages, complete bed rest is necessary. Exfoliative dermatitis resulting from hypersensitivity to the heavy metals (gold arsenic, etc.) is treated with BAL which is given by injection every 4 hours for 6 doses for the first two days, then three times a day until recovery is well advanced. If reactions occur from the

drug ephedrine sulfate should be prescribed as an antidote. Acute exfoliative dermatitis of external origin is treated with the corticosteroids and antihistaminics to relieve the pruritus.

In chronic cases, the nurse should be observant for pressure "sores," as well as furuncles, abscesses and enlarged glands. Bronchopneumonia or nephritis are the usual causes of death.

Diseases of the Hair and the Scalp

VEGETABLE PARASITIC (FUNGUS)	HYPOTHYROID ALOPECIA
DISEASES	HYPERTRICHOSIS
RINGWORM OF THE SCALP	INGROWN HAIRS
RINGWORM OF THE BLAD	ALOPECIAS
FAVUS	ENDOCRINE ALOPECIA
TRICHOMYCOSIS AXILLARIS	ALOPECIA AREATA
ALLERGIC MANIFESTATIONS OF	POSTMENOPAUSAL ALOPECIA
RINGWORM	TRICHOTILLOMANIA
ANIMAL PARASITES	SYMPTOMATIC ALOPECIA
PHIDICULOSIS CAPITIS	ATROPHIC ALOPECIA
PYOGENIC INFECTIONS OF THE	SYPHILITIC ALOPECIA
SCALP	DRUG ALOPECIAS
IMPETIGO CONTAGIOSA	ATROPHIES OF THE HAIR
FURUNCULOSIS	CANTHES
ACNE NECROTICA MILIARIA	MONILETHRIX
PERIFOLLICULITIS CAPITIS	ALOPECIA CICATRIZATA
ENDOCRINEAL DISEASES	FOLLICULITIS DECALVANS
PITYRIASIS CAPITIS SIMPLEX	TUMORS OF THE SCALP
ENDOCRINE DISTURBANCES	NURSING ASPECTS

HAIR is present on all parts of the skin except the palms and the soles, the red surfaces of the lips, the glans penis and the inner surface of the labia majora. The endocrine glands influence the distribution, the growth and the texture of the hair *

Anatomically a hair consists of a shaft, made up of an outer cortex and an inner medulla covered with an external sheath or cuticle and the bulb or root, which is supplied with an external and an internal root sheath. The bulb is attached to a papilla, which is a differentiated conical process of the corium consisting of connective-tissue fibers, pigmented cells, a vascular loop and

For a complete discussion of the anatomy the embryology and the physiology of the scalp, see Rehman, H T The Scalp in Health and Disease St Louis, Mosby 1952.

medullated nerves. The papilla forms the cells which make up the various parts of the hair shaft. Each hair is situated in a hair follicle which is an invagination of the epidermis enclosed by layers of connective tissue. The arrectores pilorum muscles have two attachments one end is attached below the opening of the sebaceous glands and the other is inserted into the cutis.

There are three types of hair the long or terminal pigmented and medullated hair found on the scalp bristles present in the ears and the nostrils and the lanugo nonpigmented and non-medullated type seen on the face in women.

Hair growth may be affected by pathologic conditions in the hair itself e.g. the presence of fungi or it may be affected through the papilla by nerve vascular, neurologic, physiologic, toxic or inflammatory disturbances. Scalp hairs have an existence of from about 2 to 4 years, after which they are replaced. Normal hair growth varies with age, site, race, endocrine function and state of nutrition and heredity. The individual hairs undergo cyclic growth with a resting phase of variable duration.

The weekly averages for hair growth (Trotter) are axillary 2.92 mm. scalp 2.73 mm. pubic, 2.09 mm. leg 1.62 mm. and arm 1.52 mm.

Hair color may be pathologically affected by various agents. Blonde shades may result from excessive sun or bleaches grayness from x ray therapy senile changes or severe psychic disturbances discoloration from Atabrine resorcinol compounds, Anthraquin brass or copper dusts.

Scalp. The skin of the scalp is dense and thick. It contains relatively few nerve endings but it has a good blood supply and incisions heal rather rapidly. Numerous sebaceous glands are present so that sebaceous cysts are fairly common in this area. Herpes zoster may affect the scalp from involvement of the auricular temporal branch of the trigeminal nerve. Lymphatic drainage is taken care of by the occipital, postauricular and deep cervical chain so that adenopathy is common in pyogenic and parasitic scalp diseases.

The normal flora of the scalp consists mainly of *Staphylococcus albus* and *Pityrosporum ovale*.

The pH of the scalp varies from 4.5 to 5.5 the acidity resulting from lactic acid secreted in the sweat. An increase in the acidity may cause local pruritus.

Examination of the Scalp Before studying the scalp all hair ornaments should be removed, and the patient placed in a good light so that the skin as well as the hairs can be carefully examined. Tongue blades can be used to separate the hairs. The extent of the alopecia, the nutritional state of the hairs, the presence or the absence of atrophy and enlarged lymph nodes should be noted.

VEGETABLE PARASITIC (FUNGUS) DISEASES

RINGWORM OF THE SCALP

Ringworm is a common contagious infection found in children. Clinically and biologically it is divided into four types (1) The common *Microsporum lanosum* type, which is acquired from infected cats, dogs or child-to-child contacts. (2) The *M. audouinii* type, which is often the cause of epidemics, is acquired by child-to-child contacts and is so resistant to local therapy that usually it must be treated by x-ray epilation. (3) *Trichophytosis* (large-spore ringworm) which is rare and usually is acquired from horses or cattle and (4) *Trichophyton tonsurans* (*T. sulfureum* or *T. crateriforme*) which causes the hairs to break off at the mouth of the follicle (black-dot ringworm) and may affect adults.

Types. MICROSPOROSIS OF THE SCALP This is the most common type of ringworm and is the result of infection with *M. audouinii* or *M. lanosum*. The incubation period is about 2 weeks. The disease starts as a localized scalliness over the occipital (clipper) area or the temporal areas. The affected areas may appear normal in *M. audouinii* infections. Then a round, oval or irregular inflammatory patch of dime size to silver-dollar size and yellowish, dirty gray to brown in color develops. The border of the lesion is sharply defined. The patch is studded with short hair stumps which have a nibbled appearance. These project from follicles which are filled with epithelial debris. The affected hairs soon become brittle, dull, loosened and thickened from infiltration with fungi; each hair has a grayish-white, frosty sheath composed of spores.*

*For an excellent review of the subject, see Beare, J. M. and Cheeseman, E. A. Their capital: Review of 1004 cases, Brit. J. Dermat. 63:165-166, 1971

TRICHOPHYTOSIS OF THE SCALP is a rare infection with *Trichophyton tonsurans* (*T. sulfuraceum* or *crateriforme*) which grow within the hair or ectothrix Trichophytions, which are found outside the hair in the sheath and the cuticle. This type of infection is characterized by (1) scaly patches that contain broken-down or corkscrew hairs curled up in the follicles, (2) black dots, which are really pigmented hair stumps, (3) pale fluorescence or none and (4) marked resistance to therapy.

Tinea kerion is a painful inflammatory lesion or a pyogenic granuloma, usually single, and may be associated with any of the ringworm fungus infections. The lesion consists of a boggy swelling composed of follicles filled with hair stumps, and the presence of small follicular abscesses. The papillae may become atrophic, with resulting permanent alopecia. In severe cases enlarged lymph nodes and constitutional symptoms are common findings. Spontaneous cure is the rule.

Etiology Ringworm of the scalp does not occur after puberty because of an increase in the fungistatic fatty acids in the sebum and the sweat. Hats, caps, brushes and combs are the usual



FIG. 178 *Tinea capitis* (kerion) in a boy of 5 (From Dr. Kathleen Riley)

fomites. Infection by *M. lanosum* is conveyed by "mangy" cats and dogs.

During epidemics, as many as 80 per cent of the cases may consist of *M. audouinii* infections.*

The Trichophytons causing large-spored ringworm which usually affects adults, are of the endothrix types (*Trichophyton crateriforme*, *T. scutigenum* or *T. violaceum*) or of the ectothrix types (*T. gypsum* or *T. ripens*).

Diagnosis. Any scaly condition of the scalp found in a patient under 14 years of age should be considered to be ringworm until it is definitely proved that it is not. However a positive diagnosis can be made only by (1) microscopic examination of the hairs in potassium-hydroxide preparations (the Hotchkiss-Mc Manus stain should be used if grease or bubbles interfere with proper visualization of the spores) (2) cultures on Sabouraud's media (infected hairs are incubated from 10 days to 2 weeks at room temperature) and (3) examination under the Wood light.

Differential diagnosis is outlined in the following table

DIFFERENTIAL DIAGNOSIS BETWEEN *M. Lanosum* AND *M. Audouinii* INFECTIONS

SOURCE	<i>M. Lanosum</i> ANIMAL	<i>M. Audouinii</i> HUMAN
Clinical	Inflammation present	Minimal
Wood light	Short luminous yellowish-green spots	None
Microscopic	Fusiform or oat-shaped spores	Rare
Cultures	Small tan colonies with yellow substratum	Grayish, white, fluffy colonies with central elevation and red substratum

T. tonsurans infections (sulfureum) may cause difficulty because they may occur in adults, Wood light examination is negative and the lesions may resemble folliculitis, seborrheic dermatitis or pseudopelade.

Course. The average case of *M. lanosum* infection is cured after from 3 to 6 weeks. Microsporosis of human origin and trichophytosis are more resistant to therapeutic measures, but 90 per cent of the cases are cured with skilled x ray therapy. Untreated,

*See Casper W. A. and Malone, J. The occurrence and control of ringworm of the scalp in the United States, New York State J. Med. 54:1645, 1954

the course of the disease is slow and progressive up to the age of puberty

General Instructions. (1) Clip hair short at home and burn cuttings. (2) Wear paper or cloth cap (bandanna kerchief for girls) and renew daily (3) Shampoo daily with green soap tincture (4) Keep scalp greased with baby oil to prevent hairs and spores from falling (5) Keep outer clothing in separate lockers to prevent infection of other children

Treatment. The type of therapy used will depend on the results of the cultural growth, the age of the patient, the extent of the infection the available therapeutic measures and the co-operation of the patient and the parents.

THERAPY FOR *M. lanosus* TYPES If the diagnosis is correct, these cases should clear up within 4 weeks with the application of the usual fungicidal ointments, viz. 5 per cent sulfur 5 per cent ammoniated mercury Asterol Quinolox or undecylenic-acid ointments.

TREATMENT OF *M. axdovini* INFECTIONS. Standard therapy is x ray epilation. The scalp is divided into 5 equidistant areas, each of which receives from 275 to 400 r The hair falls out within 18 days, with regrowth in about 3 months. Failures are due to reinfection or to unskilled therapy Retreatment with x ray must not be given before 6 months.

LOCAL THERAPY Indications for ointment therapy are (1) unavailability of x ray therapy (2) cases in infants and unmanageable children (3) lack of co-operation of parents, (4) small infected patch or few infected hairs, (5) parental objection to x ray therapy (6) x ray sickness and (7) presence of secondary infection.

Salicylanide Spergon (Doak) and copper undecylenate ointments have been effective in about 15 per cent of the cases after 3 months of vigorous treatment.

Manual epilation under the Wood light is useful for small patches

CRITERIA FOR CURE consist of two negative Wood-light examinations, one week apart, no clinical evidence of infection, negative cultures from previously affected areas and an observation period for 2 months after apparent cure. Other children in the family as well as playmates, should be examined for the presence of ringworm Combs brushes and headgear used by patients should be destroyed.



FIG. 179 (Left) *Tinea barbae*. Note the large boggy abscesses (Dr. McCuskey's case)

FIG. 180 (Right) Favus. The yellow scabrous adherent crusts are characteristic

EPIDEMIOLOGY During an epidemic, all school children should be screened with the Wood light, and all infected cases should be reported to the public health authorities and sent to a ringworm treatment center for therapy. Children if infected may continue in school so long as they are under treatment and observe the proper precautions. Barber shops should be instructed in sterilization technic.

RINGWORM OF THE BEARD

Tinea barbae is a rare suppurative folliculitis. This infection which is uncommon is limited to the male sex. Most of the cases occur in farmers and are acquired from farm animals. The disease is characterized by deep pustules and tender boggy inflammatory or impetiginous nodules. Any part of the bearded area may be affected. The fungus often found in ringworm of the beard is an ectothrix (*T. faviforme*).

Prognosis. The severe inflammatory types may involute spontaneously. Infections with *T. purpureum* and *T. violaceum* are stubborn.

Diagnosis is made by clinical signs, examination of the hairs for fungi and by cultural studies.

Differential diagnosis is as follows

Source	Seborrheic Dermatitis	Sycosis Vulgaris	Tinea Barbae
Distribution	Scalp, ears and eyebrows also involved. Look at chest and back for lesions	Usually limited to upper lip or bearded area	Upper lip rarely involved usually maxillary and submaxillary regions
Clinical lesions	Greasy scaly patches never pustular or nodular	Superficial pustules pierced by a hair; surrounding skin may be inflamed	Large deep pustules and nodules
Microscopic	Negative for fungi	Staphylococci	Fungi

Pathology shows a follicular and perifollicular inflammatory reaction. Permanent alopecia may result.

Treatment consists of the use of hot compresses of Vlemmick's solution (1:30) followed by 5% ammoniated mercury or Quinolac ointment (Squibb) foreign-protein therapy with milk or typhoid vaccine, manual or skilled x-ray epilation in the stubborn cases. Contaminated articles should be sterilized or destroyed.

FAVUS

Favus (*tinea favosa*) is a chronic infectious disease caused by the *Achorion schoenleinii* and characterized by a peculiar cup-shaped crusting with termination in atrophy. It is not a common disease and usually is acquired in childhood. Most of the cases are seen in children between the ages of 5 and 15 years. In large cities, about 75 per cent of the cases occur in recent immigrants. In inland cities, it may be found in American-born patients. Infection is acquired in childhood by direct contact, although the disease also may be contracted from animals. The scalp is the usual site of involvement and the disease often is limited to that region; however the trunk and the nails may be involved also. The disease begins as a small scaly inflammatory lesion which soon is covered with a sulfur yellow cup-shaped adherent crust. The hairs are brittle, dull and lusterless and are broken off easily. The pressure of the crusts against the papillae causes a permanent alopecia.

Diagnosis of the disease is made by the characteristic clinical appearance of the crusts and the hairs and slide and culture examinations of the infected hairs. All contacts should be examined and treated if necessary.

Differential Diagnosis. Atypical cases may be confused with folliculitis decalvans.

Treatment includes antiparasiticide ointments and lotions and x-ray epilation by a specialist. The hair should be clipped short, and local treatment should be continued for a month after apparent cure.

TRICHOMYCOSIS AXILLARIS

Trichomycosis (leptothrix) appears as yellow nodular fungus masses on the shafts of the axillary hairs. They also may be red or black. There are no symptoms and the condition is not infectious. The condition usually is discovered during an examination for some other condition. Chronic axillary hyperhidrosis with an adaptation of the organisms to an alkaline sweat predisposes to its appearance. Organisms causing the disease belong to the *Nocardia* group.

Treatment consists of removal of the hairs by shaving and applications of a 1:5,000 solution of mercury bichloride to the involved regions.

ALLERGIC MANIFESTATIONS OF RINGWORM

These include the following types:

- 1 Microsporids are banal lichenoid or squamous lesions on the face, the neck or the trunk. The lesions appear suddenly especially after x-ray epilation for scalp ringworm and disappear within three weeks. They always are associated with microsporids of the scalp. Mild constitutional symptoms may be present. Fungi are not found in the lesions but they may be present in the blood for a very short time.

- 2 Trichophytids are allergic manifestations associated with focal lesions which have become irritated. The trichophytids may be lichenoid squamous or vesicular. Trichophytin intradermal tests are usually positive. Fungi are absent in the lesions, having been killed by the allergic response of the skin.

- 3 Favids are generalized pinhead-sized scaly lesions appearing on the trunk in the presence of tinea.

4. Monilids may appear following prolonged antibiotic therapy and may be prevented by parenteral injections of vitamin B complex or antibiotics combined with nystatin.

TREATMENT These allergic lesions seldom require treatment. They usually disappear spontaneously when the primary focus is healed. If not, a weak sulfur ointment or a few x-ray treatments usually clear them up.

ANIMAL PARASITES

PEDICULOSIS CAPITIS

This infection is caused by the head louse (*Pediculus*). The disease affects the occipital region. It rarely affects the beard and the eyelashes. The parasite is brownish or grayish and is equipped with mandibles and suckers for penetration. Its nits, or eggs, are attached by a glutinous substance to the hairs and hatch in from 5 to 6 days. Most of the cases are observed in neglected children and in the aged who live alone.

Itching is a prominent symptom. The disease is characterized by excoriations, blood crusts, secondary impetigo or furunculosis



FIG. 181. Pediculosis capitis with secondary impetigo and enlarged lymph nodes.

and by postauricular and occipital adenopathy. The infection is acquired through such fomites as hats, caps, wigs, combs and brushes.

Diagnosis. The presence of nits is diagnostic of the disease. They must not be confused with dandruff which is a loose flaky scale easily removed or brushed off. Impetigo of the scalp should be regarded as being a possible secondary manifestation of pediculosis.

Treatment. All the members of the family should be examined and treated at the same time. DDT in a concentration of 10 per cent in powder form (talk, kaolin or prophyllite) is sprayed or dusted with a shaker can into the scalp and is permitted to remain for one week. The treatment is reapplied after two weeks if necessary. DDT is preferred therapy for mass treatment in schools, institutions, camps, etc. Cuprex, Eurax and Kwell are also effective local applications. Following specific treatment, the nits must be removed by soaking the hair in vinegar or 10 per cent acetic-acid solution after which the hair is combed with a fine-toothed comb. Combs and brushes used by patients should be destroyed and pillows should be laundered to prevent reinfection.

PYOGENIC INFECTIONS OF THE SCALP

There are four types of pyogenic infections of the scalp.

IMPETIGO CONTAGIOSA

Impetigo contagiosa usually occurs in children as a complication of pediculosis capitis or atopic eczema or secondary to impetigo lesions elsewhere. The lesions may be crusted or pustular.

Treatment consists of removal of the crusts and local applications of (1) 5 per cent ammoniated mercury (2) Quinolac or (3) antibiotic ointments preferably bacitracin. Frequent shampoo and general hygiene are important.

FURUNCULOSIS

Furunculosis is often secondary to poor hygiene, pediculosis or impetigo. Hot boric packs and incision when "ripe" are standard therapy.

ACNE NECROTICA MILLIARIA

Acne necrotica miliaris occurs as an eruption of very small, extremely pruritic vesicopustules. (See p. 519.)

PERIFOLLICULITIS CAPITIS

Perifolliculitis capitis abscedens et suffodiens, or dissecting cellulitis of the scalp consists of fluctuating abscesses and sinuses. The surface of the involved area is undermined by sinuses. Scarring is inevitable. Pathologically the lesions are granulomas. The disease is caused by infection with pyogenic cocci in patients with lowered resistance and occurs especially in Negroes.

Treatment of localized areas consists of surgical measures to increase drainage antiseptic dressings general measures to improve the health and autogenous vaccines Antibiotics may retard the progress, but total excision and plastic repair are often necessary for a permanent cure.

SEBORRHEAL DISEASES

SEBORRHEIC DERMATITIS

(See p 525)

PITYRIASIS CAPITIS SIMPLEX

(DANDRUFF)

Dandruff is a common superficial disease of the scalp consisting of thin easily removed scales on an otherwise normal scalp. The importance of the disease lies in the fact that it often predisposes to (1) seborrheic dermatitis and (2) seborrheic alopecia.

Etiology Pityriasis capitis is an infectious disease of unknown origin. Some observers regard it as an infection in a susceptible individual, with *Pityrosporum ovale*. The roles of the *Staphylococcus albus* and the acne bacillus are probably secondary.

Predisposing causes are family tendency and scalp neglect. The disease is very common. Practically the entire population is infected at one time or other.

Pathology The scaling which is the characteristic feature of the disease results from an excess formation of cells in the stratum corneum, due to bacterial irritation.

Histologic section reveals moderate hyperkeratosis, parakeratosis, slight acanthosis edema of the prickle-cell layer and a moderate round-cell infiltration about the sebaceous glands.

Differential Diagnosis. Seborrhea of long standing sometimes results in a festooned mildly inflammatory area at the anterior hairline which extends across the forehead. This is

known as the corona seborrheica. Severe cases must be differentiated from psoriasis, which is a patchy disease with yellowish-white thick dry scales, and also tends to involve the extensor surfaces. When psoriasis extends along the anterior hairline the resulting lesion, which resembles seborrheic dermatitis, is called the corona psoriatica.

Secondary syphilis, when it involves the forehead may consist of closely set follicular papular lesions in the same area, producing a similar type of lesion called the corona veneris.

Course. After the disease persists for several months or several years, seborrheic dermatitis usually follows from irritation and infection with *Staph. albus*. Alopecia furfuracea gradually develops in both men and women in cases of long standing. The



FIG. 187 Psoriasis capitis simplex, resulting in seborrheic alopecia

loss of hair first involves the temples and the vertex of the scalp.

Relapses are common and usually are due to reinfection and failure to continue the prescribed treatment.

GENERAL Treatment. The patient should avoid wearing tight hats and must not pick or rub the scalp. The brush should be discarded for the comb which is immersed in 1:2000 mercury bichloride solution when not in use. A towel should be placed about the neck when combing the hair to avoid auto-infection of the chest and the back. Hair oils must be eliminated.

Small doses of thyroid vitamin B complex or ferrous sulfate if indicated, may be found useful.

DIET Alcohol greasy foods, including pork, bacon, cream, butter, chocolate, cocoa and fried foods may alter the character of the sebum.

ENDOCRINE. Small doses of thyroid are often useful in both the dry and the oily types. If anemia is present, this should be corrected with iron or liver preparations.

MASSAGE. A gentle massage of the scalp is a useful procedure provided it is done daily before applying local medication. In the dry types, the stimulation of the local circulation sometimes increases the secretion of sebum.

SHAMPOO. The scalp should be shampooed every week when the scalp is dry using a superfatted or sulfated soap. The oily scalp should be shampooed daily or twice weekly depending upon the amount of oil present and the season of the year. Tincture of green soap, soap chips or a good liquid tar soap are satisfactory detergents for the greasy scalp and produce mild exfoliation of the excess horny layer and the sebum. Temporary improvement is frequent after a shampoo but further local treatment is necessary.

PHYSIOTHERAPY. Ultraviolet light therapy is useful in both types of seborrhea and produces (1) a mild local inflammatory reaction resulting in exfoliation, (2) a mild bactericidal effect and (3) stimulating action on the scalp.

LOCAL THERAPY consists of the use of lotions or ointments containing antiseptic, stimulating antiseborrheal drugs, including sulfur, salicylic acid and tar. Ointments are objectionable to both men and women especially if prescribed in a petroleum base which is non-saponifiable and does not wash out readily with soap. To remedy this situation water-absorbent bases (e.g. Carbowax

1500) are advised for scalp ointments. A thin dust cap or bathing cap is worn at night to protect the pillow and the face.

Lotions are prescribed at first, but if not effective after two weeks, ointments should be used. Liquid preparations are applied with a dropper a pledget of cotton or an atomizer. In mild cases with evidence of alopecia the following lotion is useful

(1) Resorcinol monoacetate	5
Ethyl alcohol (70%) q.s. ad	100

Selsun (selenium sulfide) and Sebzone Cream (Schering) are also effective in mild seborrhea.

Ointments are rubbed in at bedtime with a toothbrush or with the fingers, which should be protected with gloves or a finger cot. One of the following drugs may be incorporated in the scalp ointment

(1) Precipitated sulfur	3 to 6%
(2) Salicylic acid	1 to 6%
(3) Ammoniated mercury	3 to 6%
(4) Liquor pitch carbonis	3 to 10%
(5) Eucnaol	3 to 6%
(6) Hydrocortisone combined with oxytetracycline or neomycin	1 to 2.5%

An ointment that has been useful in the author's practice is the following

Precipitated sulfur	60
Salicylic acid	30
Oxyquinoline sulfate	10
Carbowax 1500 ad	1000

§ Massage into scalp every night.

Prognosis. No permanent cure is known, but the condition can be kept under control by persistent treatment and attention to scalp hygiene and the general health.

ENDOCRINE DISTURBANCES

HYPOTHYROID ALOPECIA

Hypothyroid alopecia with accompanying harsh dry and brittle hair is one evidence of endocrine disturbance. It may be associated also with a dry skin brittle nails, a low basal metabolism rate and constitutional symptoms. The alopecia is diffuse. Thyroid medication is usually effective but consultation with an internist is advisable.

HYPERTRICHOSIS

Superfluous hair is an excessive growth of hair on areas normally covered with lanugo hairs. This abnormal hairiness may be a clinical problem. Most of the cases occur in women and usually are limited to the upper lip or the chin. In extreme cases, the growth of hair is generalized. Even in mild cases, an anxiety neurosis or an inferiority complex may be present, resulting in neurotic excoriations from attempts at pulling out the hairs.*

Etiology The following factors and conditions may be associated with hypertrichosis: (1) heredity with increased susceptibility of the follicle to normal hormonal stimuli, (2) *spina bifida*, (3) large pigmented moles, (4) acne complex, (5) Cushing's syndrome, (6) endocrine disturbances especially pituitary disorders and artificial and natural menopause, (7) arrhenoblastoma of the ovary and adrenal tumors (with adrenal cortical hyperfunction), (8) atavism, (9) injections of testosterone in females, (10) corticotropin (ACTH) and (11) porphyria.

Diagnosis. Masculinizing pathologic processes first must be ruled out by clinical evidence (timbre of voice, skin texture, fat padding status of clitoris, etc.) evaluation of ovarian function determined and racial or genetic factors recognized. Assay of the 17 ketosteroids is not a reliable method of determining extensive androgen production.

Treatment. ELECTROLYSIS AND SURGICAL DIATHERMY are the only methods of treatment offering permanent cure. Mechanical removal by shaving, manual epilation or plucking or the use of waxes and pumice are temporary measures. The effect of shaving on the rate of regrowth of hair is a disputed matter. It should be advised only for those women with thick growths. The hairs may be removed chemically by the use of pastes containing barium, strontium or calcium sulfides or the more elegant calcium thioglycollate creams.

BLEACHING which renders the hairs less conspicuous, is performed by washing the hair with soap and water then applying weak ammonia water for five minutes followed by hydrogen peroxide (20 Vol. strength) for 20 minutes.

HIGH FREQUENCY DIATHERMY is a rapid and satisfactory method.

For an excellent discussion of the subject, see Illustrations, chap. 20, in Savill, Agnes. *The Hair and Scalp*, ed. 4. Baltimore: Williams & Wilkins, 1952.

ELECTROLYSIS by galvanic current, although slow is the safest method. The single-needle method is preferable. (For details of technic see p 609)

SHAVING with the electric razor is advisable in the extensive cases. The skilful use of toilet pumice stone may produce satisfactory results though temporary

ESTROGENS in large doses may be tried for extensive cases in which there is no evidence of malignancy but results are generally unsatisfactory

INGROWN HAIRS (PILI INCARNI)

Ingrown hairs are the result of an anatomical defect of the follicle, with the production of one or more painful papules. Inflammation is minimal, and pus is rarely present. The lesions may occur anywhere on the bearded area. In Negroes the lesions may become keloidal

Etiology The condition may be caused by (1) oblique follicles, causing the hairs to grow parallel to the surface, (2) close shaving, resulting in hairs growing through the wall of the follicle and (3) the presence of curved hairs which grow back into the skin.

Differential diagnosis must be made from traumatic folliculitis and syccosis barbae.

Prognosis. The condition is usually persistent.

Treatment. Use of the electric razor and removal of the hairs with the epilating forceps usually controls the condition.

ALOPECIAS

The alopecia may be patchy or diffuse temporary or permanent. The various types are classified as follows

1 Alopecia associated with atrophy burns and scalds lupus erythematosus pseudopelade pyrogenic infections syphilis, tuberculosis and cancer x ray dermatitis.

2 Alopecias associated with inflammatory diseases of the scalp tinea capitis and kerion seborrheic dermatitis and contact dermatitis from permanent waving etc.

3 Alopecias without obvious changes in the scalp alopecia areata congenital alopecia masculine hereditary alopecia symptomatic alopecia trichotillomania trichorrhesis nodosa alopecia associated with lymphoblastomas.



FIG 183 Alopecia (*Upper left*) Congenital alopecia (*Upper right*) Seborrheic alopecia (*Lower left*) Alopecia areata (*Lower right*) Symptomatic alopecia, arnica exfoliative dermatitis (Dr Leon Goldman case)

THE CONGENITAL TYPE is rare. It is due to lack of development of the hair follicles and often is associated with other developmental defects. There is no cure.

THE INFANTILE TYPE is a temporary alopecia, occurring in many babies soon after birth affecting the parietal and the frontal areas in both sexes. Shedding of the hair may continue gradually until the end of the first or the second year when complete replacement takes place.

THE SENILE TYPE is a diffuse alopecia which usually is associated with graying of the hair and other signs of senility (loss of teeth, wrinkled skin etc.) The time of its appearance varies in different individuals. There is no successful treatment.

PREMATURE ALOPECIA develops between the ages of 18 and 25 years. It is limited to males and appears in the frontal and the vertex regions. Most cases have an hereditary background however some are due to seborrhea disturbance of the androgen estrogen ratio, poor hygiene or alkaline soaps. If the patient appears depressed or develops an anxiety neurosis, psychiatric consultation may be advisable.

Treatment given early may prevent further loss. Thyroid substance if indicated is often of value. The daily wetting of hair with water and the use of harsh brushes are contraindicated. Ultraviolet light therapy in erythema doses appears to have a stimulating effect on the viable hair follicles.

The author uses the following form for history taking in the alopecias

HISTORY FORM FOR ALOPECIAS

SCALP QUESTIONNAIRE

- 1 How long has your hair been falling?
- 2 Over what part of the head does the hair loss seem to be the greatest?
- 3 How much do you brush your hair?
- 4 How much hair remains on your comb each time you use it?
- 5 Is your hair dry or oily?
- 6 Do you have dandruff, scabiness or itching of the scalp?
- 7 Do you use pomade, fat, lacquer etc?
- 8 How often do you shampoo your hair and what brand do you use?
- 9 Have you had a permanent wave recently? What effect did it have on your hair?
- 10 Is your skin dry or oily?
- 11 At what age did grayness appear?
- 12 Do the finger nails break off easily?

- 13 Did you have an infection in your throat, teeth, before your hair fell out?
- 14 Have you been ill or had a fever or operation before the hair fall?
- 15 Have you been taking sulfa, injections or other medicines?
- 16 Have you been on a strict diet of late?
- 17 Have you had a baby recently?
- 18 Do you feel tired all the time?
- 19 Have you suffered from shock or been under an emotional strain of late?
- 20 Has anyone in your family had the same trouble?
- 21 Are you really worried that you might be bald?
- 22 Do you ever have a crawling sensation in the scalp?

SEBORRHEIC ALOPECIA

Seborrheic alopecia is associated with seborrhea of long standing. It begins at an early age during puberty. The disease is usually diffuse, starting at the vertex.

Treatment. It is necessary to shampoo the scalp weekly with tincture of green soap when it is oily or every two weeks, if dry preferably with a superfatted or castile soap. In the early stages, ointments containing sulfur, resorcinol and salicylic acid or Selsun lotion are useful. Later "hair tonics" containing one or more of the following may be used: tincture cantharides, tincture capsicum, hydroxyquinoline, spirits of formic acid, chloral hydrate, quinine bisulfate, pilocarpine, resorcinol, sulfur, salicylic acid or tar.

Resorcinol monoacetate	4-8.0
Mercury bichloride	0.2
Salicylic acid	4-8.0
Glycerin	8.0
Ethyl alcohol (70%) q.s. ad	240.0
M. ft. lotion	

Use for oily seborrhea.

Resorcinol monoacetate	4-8.0
Mercury bichloride	0.2
Salicylic acid	4-8.0
Castor oil	8.0
Ethyl alcohol (70%) q.s. ad	240.0
M. ft. lotion	

Use for dry seborrhea.

Prophylaxis. Most cases are preventable if the dandruff (pityriasis capitis) is treated properly. Hygienic care of the hair and the scalp includes avoidance of tight hats, use of clean combs and brushes, daily brushing of hair, daily massage of scalp and removal of all soap from the scalp after the shampoo.

ALOPECIA AREATA

Alopecia areata is characterized by a sudden loss of hair occurring in susceptible individuals in one or more circumscribed patches and usually involving the scalp. There is no visible evidence of inflammation. This fact is of diagnostic importance. The patches, if multiple, are often of various sizes from that of a dime to that of a silver dollar; they are oval, round or irregular; the surface is smooth and shiny. The beard, the eyebrows, the eyelashes or the axillae may be affected also. Sometimes the entire scalp is involved, as in alopecia totalis; or in rare cases, all the hair on the body may fall out, as in alopecia universalis.

In 10 per cent of the cases the bald patches may be preceded by or associated with paresthesias or neuralgias in the affected area.

Etiology is vague. There is often a history of shock or emotional strain. Other contributory causes are errors of refraction, nervous exhaustion, pregnancy, malposed teeth, surgical operations, spinal anaesthesia and neuroses. Occasionally the disease is preceded by local trauma. Evidence regarding an endocrine basis for the disease is conflicting. The cause probably acts through the vegetative nervous system.*

Pathology of the disease consists of a disturbance of the nutrition of the papillae with or without atrophy of the follicles, and a mild inflammatory reaction in the upper corium.

Differential Diagnosis. The first thing to observe is the presence or absence of atrophy. In children, tinea capitis should be ruled out by Wood-light examination and cultures and microscopic examination of the hairs in or about the patch. Trichotillomania is more diffuse and the hairs are broken off at different levels. If atrophy is present (thin, shiny, tense skin) the diagnosis is apt to be cicatricial alopecia or an old lupus erythematosus.

*For more complete details of etiology see Tobias, Norman: Alopecia areata, *Postgrad Med* 15:50-53, 1954.

Prognosis. Most observers feel that the disease will be cured when Nature decides that it shall be cured. Generally the prognosis is good but the hair rarely grows in before three months. However, in patients over 45 years of age, there is a possibility that the condition may be permanent.

Hair regrowth is indicated by the following consecutive signs: darkening of the follicles, the appearance of fuzz, depigmented and finally pigmented hairs.

Treatment. The only important form of therapy is the production of hyperemia by ultraviolet light, 7 per cent tincture of iodine, liquid phenol, tricresol or Cutler's solution (equal parts of tincture iodine, chloral hydrate and phenol). Phenol or tricresol is carefully applied to each patch if not larger than a silver dollar until it turns white, after which it is neutralized with alcohol. Office applications once a week are sufficient. Small doses of thyroid may be prescribed as collateral therapy if the basal metabolism rate is below normal. Daily rest periods are important to conserve nerve energy. Hydrocortisone which must be used for at least 10 weeks or longer may be effective in the extensive types, but the hairgrowth may be temporary.

POSTMENOPAUSAL ALOPECIA

Postmenopausal alopecia is usually resistant to treatment, although further loss of hair may be prevented by the use of estrogenic ointments, thyroid medication and multiple vitamins. Anxiety neuroses should be counteracted by discussion and a common sense approach. Artificial restorations may be satisfactory to the patient.

TRICHOTILLOMANIA

Trichotillomania is a condition characterized by an involuntary plucking or rubbing of hairs. It is found in psychoneurotics, cocaine addicts and acrophobics who imagine that the hairs are infested with parasites. In children the cause may be tension as a result of a domineering parent or a frenzied attempt to escape an unavoidable situation.

Diagnosis is usually simple since the patient admits that he pulls out the hairs. The presence of hairs unequal in length but too short to be plucked is important. The absence of fluorescence under the Wood light, normal hair stumps, normal resistance to epilation and absence of scaling rule out ringworm.

Therapy The hair should be cut very short. Painting the affected areas with collodion may be effective temporarily. An attempt should be made to direct the energy of this neurotic behavior into other channels. In persistent cases, a psychiatric consultation is advisable.

SYMPTOMATIC ALOPECIA

Symptomatic alopecia is a loose term for loss of hair occurring during chronic and acute illnesses, including severe anemia, scarlet fever, typhoid fever, influenza, pregnancy, postoperative crises, angina pectoris and tuberculosis. Alopecia may occur in those on strict diets with no supplemental vitamin intake. Nail growth may also be affected.

Prognosis. Regrowth can be assured in most cases.

Treatment. Stimulating lotions (see p. 497) may accelerate the alopecia by increasing the loss of loose hairs. Weekly ultraviolet therapy, multiple vitamins and encouragement are necessary adjuncts.

ATROPHIC ALOPECIA

This includes those types secondary to such conditions as x-ray dermatitis, lupus erythematosus, favus, kerion, burns, sarcoidosis, metastatic carcinoma, leprosy and gummas.

Treatment is unavailing.

SYPHILITIC ALOPECIA

This is a type of symptomatic alopecia limited to the early stage of syphilis. There are two varieties: the diffuse type usually mild, which may occur in any chronic disease; and the so-called "syphilitic alopecia." The latter type usually affects the posterior part of the scalp but the beard or the eyebrows may be involved also. A follicular or pustular syphilid often is present on the trunk. The patches have a "moth-eaten" appearance and are noninflammatory. Sections show spirochetes in the follicles and around the papillae. Regrowth of the hair occurs (See Fig. 122.)

DRUG ALOPECIAS

Thallium acetate, aminopterin, overdosage with vitamin A and the thioracils may cause alopecia.

ATROPHIES OF THE HAIR

CANITIES

Canities, or graying of the hair, consists of two types. (1) premature canities, due to hereditary influences, nervous disorders, malnutrition or the use of strong alkaline soaps, and (2) senile canities. When advising patients regarding the dyeing of the hair it is well to remember that about 10 per cent of all people are hypersensitive to paraphenylenediamine dyes. The best treatment for gray hair is "to admire it." Para-aminobenzoic acid and calcium pantothenate are of no value.

MONILETHRIX

Beaded hair is a rare condition with an hereditary and familial background. It is noticed first in infancy or early childhood, at which time the scalp shows diffuse alopecia and follicular hyperkeratosis of the occipital region. Close inspection reveals alternate light bands, the atrophic constricted areas, and dark bands, the zones of normal growth. The hairs break off at the inter nodes and from friction. Differential diagnosis is mainly from hypotrichosis, keratosis pilaris and trichorrhexis nodosa. The physician should impress the patient with the hereditary nature of the disease.

Treatment. Hydrocortone ointments or oral corticosteroids may be used on a trial basis.

ALOPECIA CICATRIZATA

Alopecia cicatrizata (pseudopelade) is a rare type of permanent alopecia, consisting of discrete oval, round, or irregular "onion skin" shiny multiple atrophic areas which resemble "foot prints in the snow." The islands of alopecia are separated by tufts of coarse hair. At the borders of the patches, twisted atrophic or hyperpigmented hairs may be found. There is definite atrophy present with destruction of the hair follicles. The onset is very gradual and usually appears first on the vertex. There are no inflammatory signs, crusts, pustules or broken-off hairs present. A pink or violaceous border sometimes surrounds the periphery of the lesions.

Etiology. The cause is unknown. The disease is more common in males from 30 to 60 years of age. Some cases are associated with lichen spinulosus.

Differential diagnosis is from alopecia areata folliculitis decalvans, metastatic carcinoma and atrophic lupus erythematosus.

Prognosis is poor

Treatment. A complete physical examination should be made to discover focal infections or metabolic disease. Local therapy consists of ultraviolet light exposures, massage and ointments containing tar and sulfur. Maximum doses of vitamins A and E alternating with thyroid extract (if indicated) may be beneficial in limiting the disease.

FOLLICULITIS DECALVANS

Folliculitis decalvans is a disease of the scalp in which there are pen-sized to palm-sized areas of atrophy with a peripheral folliculitis, pustules and crusts. In the early cases, the closely aggregated folliculitis resembles sycosis vulgaris. In the late cases the central depressed scarring and the peripheral folliculitis are characteristic. There are alternate periods of activity and quiescence. The disease probably is due to the *Staphylococcus aureus*.

Differential diagnosis is from lupus erythematosus, in which atrophy also is present, but follicular pustular lesions are absent. In children, favus and tinea tonsurans should be ruled out.

Treatment. Ultraviolet light exposures help to control the condition. Autogenous vaccines and antibiotic ointments are useful in refractory cases.

TUMORS OF THE SCALP

These tumors are of two types: the benign and the malignant.

The benign type includes sebaceous cysts (see p. 362) neurofibromata and verrucae which are usually of the digitate type.

The primary malignant tumors are basal-cell cancer, squamous-cell cancer (often arising from sebaceous cysts) and melanoma.

The secondary malignancies include hypernephroma, squamous-cell cancer and sarcoma.

NURSING ASPECTS

Preparations for examination of the scalp depend upon the tentative diagnosis made by the physician. In any case a clean towel should be placed about the patient's neck, all ornaments should be removed from the scalp, and the patient should be seated so that unobstructed daylight is thrown upon the area.

The physician must determine whether the disease affects the hairs alone or the hair follicles or the skin or the subcutaneous tissues. Furuncles and millaria usually affect nursing infants. Impetigo furuncles, pediculosis and ringworm are found in children. dandruff seborrheic dermatitis, alopecia areata and premature baldness affect young adults while cicatricial and menopausal alopecias, tumors of the scalp and psychosomatic disorders appear in older individuals.

The following instruments and supplies should be available: tongue blades to separate the hair, magnifying lens, clean microscopic slides and cover slips, hair forceps, watch glass, alcohol, 20 per cent potassium-hydroxide solution in a dropper bottle, Wood light and set of instruments for biopsy.

Ringworm of the Scalp. All the children under the age of 14 in the family must be examined under the Wood light. The scalp must be shampooed first with tincture of green soap to remove all foreign matter, scales and ointments. Samples of the positive fluorescent hairs are pulled out with a sterile forceps and placed in a clean watch glass containing 70 per cent alcohol. Implantations are made carefully on 2 slants of Sabouraud's agar and the remainder are placed on a glass slide for microscopic examination. While waiting for the organisms to grow which requires about 10 days, a semi-isolation procedure must be followed. A skull cap made of clean linen or a stocking should be worn at all times. The hair should be clipped at home; the clippings should be burned and the scalp should be shampooed daily. An ointment containing 5 per cent ammoniated mercury should be massaged into the scalp daily. At the end of the 2 week period the scalp should be examined again under the Wood light, and the cultures should be inspected. If the growth reveals the organism to belong to the human type (*Trichophyton tonsurans*) the physician may decide upon manual or spot epilation (small patches in girls), local application of special fungicidal ointments (when the parents object to x ray epilation) or the use of the x ray to cause a therapeutic alopecia of the scalp.

The hair should be clipped before x ray epilation, and a mild sedative may be given to restless children. The nurse should assist the physician in marking the scalp and measuring the distances between exposures. Sandbags and lead foil to protect the adjacent areas must be available. Relatives or friends should not be permitted to be in the room while the treatments are given.

An adhesive-plaster dressing usually is applied to the scalp to facilitate removal of the loosened hairs. After epilation a child may be permitted to attend school, depending upon the regulations of the local health department. Within the classroom, infected children should be seated as far from the rest of the class as possible. Supervision should be provided on the school play grounds to prevent head-to-head contacts. Children with scalp ringworm should be admitted 10 minutes later and dismissed 10 minutes earlier than the regular group.

Pediculosis Capitis. A thumb forceps should be available to demonstrate the parasite to the patient or the parents, but this must not be done if the patient is of the fastidious or the tense type. School nurses should examine all the members of the class when the disease appears in the classroom.

Alopecia. Hairs usually are examined microscopically in order to study nutritional defects. The nurse should be cautioned regarding burns when giving ultraviolet light treatments to patients with bald heads.

Diseases of the Sebaceous Glands

INFLAMMATORY DISEASES

COMEDONES

ACNE VULGARIS

THE ACNEFORM ERUPTIONS

ACNE NECROTICA MILIARIA

ROSACEA

SEBORRHEA

SEBORRHEIC DERMATITIS

CYSTIC DISEASES

BENIGN SEBACEOUS ADENOMA

SEBOCYTOMATOSIS

NURSING ASPECTS

Anatomy The sebaceous glands are closely associated with the hair follicles except on the vermillion border of the lips, the penis, labia minora and the nipples, where they exist independently. The largest sebaceous glands are found on the face the midline of the trunk and the back. Under certain conditions they may act as a portal of entry for infection. Seborrheic disorders occur commonly in those areas where sebaceous activity is present.

SEBUM which is the natural lubricant of the hair is an oily substance, consisting of fats, fatty acids, cholesterol, triglycerides, squalene and water. Its fatty-acid content is a factor in the self sterilizing power of the skin.

SEBACEOUS ACTIVITY which occurs physiologically during puberty when seborrheic dermatitis and acne make their appearance, is characterized by increased oiliness of the scalp and the skin, the presence of comedones and dilated pilosebaceous orifices. At puberty sebum of the sebaceous glands of the scalp contains a high concentration of saturated fatty acids which have a selective fungistatic and fungicidal action on *Microsporon* ringworm.

The sebaceous glands are subject to inflammatory cystic and nevic diseases.

INFLAMMATORY DISEASES

COMEDONES (BLACKHEADS)

Comedones are dirty-gray or dark brown, slightly elevated lesions obstructing the mouth of a sebaceous follicle.

The usual locations are the forehead the sides of the nose the midchest, the upper back, in the shell of the ears and in the external auditory meatus. They consist of dried sebum, epidermal cells from the lining of the pilosebaceous follicle microbacilli and *B. acne*. When extruded they appear to be a waxy mass.

Etiology. Comedones appear in the first stages of acne. In juvenile acne they are the predominating lesion. They may be present also in some types of superficial scars and atrophic lesions and may assume a nevus form (*nevus comedonicus*). Giant lesions may be seen in senile patients.

Sequelae. Secondary infection with the *Staphylococcus* and *B. acne* results in the typical acne pustule. If the follicular mouth of an active sebaceous gland is obstructed by the comedone a sebaceous cyst results.

Treatment. After thorough washing with soap and water the comedone should be expressed gently with a comedone extractor. Bruising should be avoided. The site should be dabbed with a little alcohol afterward. If the comedone fails to extrude, it may be softened with Sebanil (Texas Pharmacal Co.).

ACNE VULGARIS

Acne vulgaris is a chronic inflammatory disease of the sebaceous glands, characterized by comedones, papules, pustules and scars. The disease is very common in both sexes.

Acne is closely related to puberty and makes its appearance between the ages of 12 and 18 years. The condition is usually associated with an oily skin which often is inherited. The face, the shoulders, the midline of the upper chest and the upper third of the back and arms are affected commonly.

Types. **PREADOLESCENT ACNE** consists primarily of follicular lesions on the forehead. The onset of the eruption is gradual although in some cases it appears somewhat suddenly over a period of a few weeks. The patient first notices an increased oiliness, a dirty cast to the skin and the formation of comedones on the nose, the forehead or the chin. The large pores are due to stretching from plugging of the pilosebaceous openings by comedones, loss of tone of the arrectores muscles and hyperkeratinization.

PUSTULAR ACNE. Pustules predominate in this type and may be either superficial or deep. Involution is always followed by more or less scarring.



FIG. 184 (Left) Severe acne vulgaris with numerous pustules and scars (Dr. C. C. King's case) FIG. 185 (Right) Acne with neurotic excoriations

PAPULAR ACNE consists of an eruption of hard, indolent papules and comedones. The condition is common in males with coarse oily skins. Papular types may become pustular during the hot, summer months or from the prolonged influence of a tropical climate.

INDURATED ACNE is characterized by deep subcutaneous abscesses caused by the staphylococcus.

CYSTIC ACNE consists of indolent cysts of various sizes which usually are intermingled with papules and pustules. Milla are present also. Dried sebum obstructs the mouths of the sebaceous glands producing the characteristic cysts. They occur commonly beneath the ramus of the jaws and the sides of the neck.

SEBORRHEIC ACNE is associated with a seborrheic soil. The greasy scales of seborrheic dermatitis may be found in the scalp. The skin of the face is greasy and covered with numerous comedones and small acuminate follicular seborrheids. This type of acne usually is limited to the upper half of the face and involves the forehead especially.

CHIN ACNE is an indolent pustular and indurated type of acne limited to the chin. Usually it occurs in women. In most of the

cases there are associated menstrual disturbances. The condition tends to flare up before or after each menstrual period.

EXCORIATED ACNE OF YOUNG GIRLS. In girls with a neurotic disposition the occurrence of facial blemishes sometimes produces an inferiority complex. Maternal anxiety may stimulate undue attention to a banal facial condition. There is an irresistible desire to pick and to squeeze the lesions, so that numerous excoriations occur.

KELOIDAL ACNE. Keloids may occur in the deep pustular type during or after resolution of the disease. Small keloids are not unusual in the pustular type.

Etiology. While the disease is essentially microbic in character the following predisposing factors prepare the soil for infection of the sebaceous glands: age, puberty and adolescence, constipation, menstrual disorders, focal infection, seborrhea of the scalp and specific hypersensitivity to certain foods viz. chocolate and milk.

The exact role of the gonadal glands in acne has not been determined although the balance of the androgenic and the estrogenic hormones in the blood is disturbed in both sexes. The disturbed balance may not only predispose to comedo formation but also through changes in the pH, so alter the natural defenses of the skin as to render it more vulnerable to the normal bacterial flora.*

When acne persists after the age of 35 the following causes should be investigated: iodine or bromine sensitivity, ovarian deficiency, sexual incompatibility and testosterone therapy.

As to psychosomatic factors most patients, or at least their parents, are much concerned regarding the disfigurement and the possibility of scarring. Wittkover believes that in acne there is an arrest in the emotional and the psychosocial development at the stage of puberty in addition to the biologic factors.

Bacteriology and Pathogenesis. It is probable that the staphylococcus and the acne bacillus play a secondary role in the production of acne lesions. In the confusion that exists regarding the exact pathogenesis of acne the following theories have been elaborated:

*Haley and T. V. The Androgen hormones in dermatology. Brit. J. Dermat. 63: 191.

(1) The fatty secretions of the follicle can be split by bacteria into choline and other end products which have irritating properties.

(2) The follicles in acne subjects are hypersensitive to androgen and react with hyperkeratosis.

(3) Pressure of the comedone, acting as a foreign body may reduce the resistance of the perifollicular tissues to infection.

Pathology There is a perifolliculitis and an endofolliculitis of the lanugo hair follicles and the adjacent sebaceous duct. In pustular cases necrosis of the center of the lesion may occur. Occasionally giant cells are found in the cellular infiltrate.

Diagnosis is relatively easy and is based on the following points (1) age (2) localization (3) presence of comedones and (4) history of the disease.

The record of the history and the examination of an acne patient should contain the following information (1) type and amount of previous treatment (2) duration and location of eruption (3) presence of superfluous hair and scars (4) hypersensitivity to drugs and specific foods (5) presence of endocrine disorders (6) examination of scalp for seborrhea (7) menstrual disorders (8) focal infections (9) constipation (10) facial hygiene and cosmetics.

Differential diagnosis must be made from rosacea and iodide or bromide eruptions. Close inspection will distinguish acne from adenoma sebaceum. Testosterone may produce an acneform eruption in females. Tumors of the adrenal cortex may be associated with acne, altered habitus, sexual precocity, ecchymoses and polycythemia.

The appearance of acne lesions in patients taking ACTH or cortisone is an indication of overstimulation. The dosage should be decreased if possible.

Prognosis Untreated acne may persist up to the age of 35 or longer and may leave more or less scarring. X-ray therapy will cure 90 per cent of the cases although 30 per cent will have occasional relapses. Acne associated with dysmenorrhea is difficult to cure unless the menstrual disturbance is corrected. If acne persists into married life, a lack of emotional or physical satisfaction may be causative. While most types of acne improve during the summer months, excessively humid weather may stimulate the production of pustular lesions. Recurrences may be avoided sometimes by keeping the scalp free of dandruff, weight control, low

carbohydrate diet, eliminating deleterious psychosomatic influences and frequent sunlight exposures of the entire body.

Treatment. Acne always must be considered as being an important disease. Sensitive youth justly regards acne as being an impediment both in the social and the business worlds. Untreated acne may continue for several years and usually leaves permanent disfiguring scars. The principles of therapy are (1) prevent scarring by using antibacterial agents (2) correct overactivity of the sebaceous glands by astringent local therapy or x-ray (3) overcome hyperkeratosis of the follicles by peeling agents and removal of follicular irritants (4) stimulate local phagocytosis with ultraviolet light (5) drain cysts and abscesses (6) modify chemistry of the skin by dietetic control and (7) correct hormonal imbalance by small doses of estrogens if indicated.

The majority of cases of superficial papular acne will respond satisfactorily to proper local therapy. About 25 per cent may require x ray therapy. Acne surgery and antibiotics are necessary in the deep pustular types while the cystic types require electrocoagulation rather than local therapy.

HYGIENE. The face should be washed thoroughly with warm water and soap at bedtime after which comedones should be expressed and local applications should be applied as directed. Those with dry or delicate skins should use superfatted soaps. A brisk facial massage with a medium-hard brush and tincture of green soap is beneficial in the oily types. In all cases of acne the scalp should receive the proper attention. Facial massages and oil shampoos, hair oils and greases are contraindicated.

COSMETICS. All cosmetics should be removed with soap and warm water at bedtime. Face and massage creams, and cream rouges are not permitted. However during the cold months one of the tragacanth lotions may be prescribed as a powder base and an emollient. Colloidal sulfur (2 per cent) may be added to the face powder. To conceal the acne lesions and scars partially during the day one of the flesh-colored lotions may be employed.

LOCAL TREATMENT. Comedones should be expressed gently with an extractor, a grooved spoon containing a hole which is pressed over the lesion. When numerous comedones exist, they may be softened before expression by hot boric acid compresses or by applying a 4 per cent solution of triethanolamine.

Acute cases with numerous pustules and abscesses should be treated with hot boric-acid poultices or Vlemingck's solution compresses (1 tsp in 1 glass of hot water) and antibiotics before specific therapy is used.

Local Applications The standard lotion prescribed commonly is lotio alba, or White Lotion N.F. 4 per cent, which has exfoliating astringent and antiseborrheal properties. To be of value it must be prepared freshly with fresh ingredients. Kaolin (10 per cent) colloidal sulfur (from 3 to 5 per cent) resorcinol (from 1 to 3 per cent) or betanaphthol (from $\frac{1}{10}$ to $\frac{1}{2}$ per cent) may be added to increase the effect. Treatment is started with a 6 per cent mixture and the strength is increased gradually to 16 per cent if tolerated.

Lotio alba—

Zinc sulfate	6 to 16
Potassium sulfuret	6 to 16
Rose water to make	100

The following commercial acne lotions are available although they are not as effective as freshly prepared lotio alba

1 Sulfur-resorcin products Resulin (Abray) Sulforcin (Texas)

2 Sulfur lotions Polythionate (Upjohn) Dermasulf (Smith) Collo-sul (Crookes) Intraderm Sulfur (Wallace) Lotio Albulfa (Doak)

3 Powders to be made into lotion Pronac (Fougere)

4 Estrogen lotions Acnesterol (Dermik)

Surgery Since acne is a disease with a marked tendency to scarring most patients resent any measures which will add further disfigurement. While it is not advisable to open fresh pustules or cysts, indolent ones should be incised carefully with a sterile sharp-pointed knife and an antiseptic wet dressing should be applied.

TREATMENT OF ACNE SCARS. Results by experienced operators have been uniformly good. The best methods are the (1) sand paper scarification technique and (2) surgical planing with rotary steel wire brushes.*

SYSTEMATIC TREATMENT Vaccines Indications bacterial allergy (severe inflammation) Fair results have been obtained in some cases of pustular acne and cold abscesses with Staphylo-

*For details, see Eller J. J. Removal of pitted acne scars by surgical planing, New York State J. Med. 54: 1166-1169, 1954

coccus Ambotoxoid (Squibb) or staphylococcus serobacterina.

Streptokinase Streptodornase solutions (Varidase Lederle) are effective in healing sinus tracts in the deep pustular types. The solution is used to irrigate the lesions 2 or 3 times weekly.

Hormone Therapy Indications for estrogens Intractable acne in both sexes menstrual exacerbations masculine secondary characteristics present in females Estrogens should not be used routinely They are worthy of a trial in cases which resist local therapy and in cystic and indurated types. The dose depends upon individual tolerance and disturbance of the normal menstrual cycle should be avoided The response in male patients has been disappointing. Estrogens should not be used in patients under 18 years of age. Topical estrogens have not been effective in my practice although some investigators claim good results.

Thyroid may be used in resistant cases (1 gr daily for 3 months) in which there is clinical evidence of hypothyroidism and a low basal metabolism rate Resistant pustular types may respond to triple sulfonamides multiple vitamins and a high protein diet and an extended vacation at a seashore resort with graduated exposures to the sun.

Vitamin Therapy Maximum doses of vitamin A (50,000 units twice daily) may be useful in the follicular dry types of acne A trial period of 2 months is necessary to observe the effect. Poly vitamin preparations or Ascorbic acid (50 mg t.i.d.) may fortify local therapy in asthenic patients poor eaters and those who are underweight.

Physiotherapy Ultraviolet light therapy is of definite value in all superficial and juvenile types of acne in those with dry or delicate skins and in those who have had a maximum x-ray dosage. In cases where pitted scars remain after x ray treatment, surgical dermal abrasion is often productive of good results.

X-ray Therapy Indications Continuous activity persistent oilness failure of topical therapy evidence of scarring deep indurated lesions When properly used by dermatologists x ray therapy has resulted in a cure in about 90 per cent of the cases. In the pustular indurated and cystic cases in those with oily skins, x ray treatment is of great value It should not be used in juvenile and menstrual acne or in the treatment of patients with delicate skins or florid complexions. From 10 to 20 per cent of the cases may require follow up treatment after a year or more of freedom from relapse

Antibiotics Pustular types and those cases in which a low-grade infection causes resistance to the usual therapeutic measures often respond to Terramycin or Aureomycin (100 mg t.i.d.) for 2 week or 3-week periods.

Dietary Management While the role of the diet in the causation of acne has been exaggerated certain foods aggravate eruptions in some cases. This may be determined by the history. As a general rule the following foods should be eliminated: chocolate and its products, malted milks, nuts, iodized salt gravies and all fatty and indigestible foods. The heavy milk drinkers should be limited to skim milk.

PSYCHOTHERAPY Indications: excoriated acne with severe mental depression. These patients, usually females, can be handled by the physician by offering a good prognosis, sympathy, tact and understanding.

The author issues the following instruction sheet to his patients who suffer from acne *

INSTRUCTIONS FOR THE PATIENT WITH ACNE

I Care of Skin

- 1 Wash the face with the special soap using hot water for 5 minutes, then blot dry.
- 2 Apply the lotion with a folded handkerchief (not cotton or fingers).
- 3 In the morning, wash off with your regular soap.
- 4 Never squeeze a pimple or blackhead. You might damage the skin.
- 5 If you have a few ugly blemishes, you can purchase Covermark to hide them during the day.
- 6 Never use creams on the face.
- Avoid using the mirror more than necessary.

II Diet

- 1 Not more than 1 glass of milk daily. Skim milk may be taken anytime.
- 2 No ice cream or chocolate candy—sorbets may be taken.
- 3 Fresh fruit should be eaten before retiring.
- 4 Avoid "hot dogs" and chili, nuts and popcorn.
- 5 Avoid pancakes, French toast and waffles.
- 6 No cola drinks permitted, all other carbonated drinks are satisfactory.
- 7 Avoid fried foods, pork and rich gravies.
- 8 Avoid iodized salt, Bromo Quinine and Bromo-Seltzer.

III Care of Scalp

- 1 The scalp should be washed at least once a week.
- 2 If you have dandruff you will be given a special preparation.
- 3 Oil shampoos are to be avoided.

For further details in the management of acne see *Acne vulgaris*, pp. 9-39, by Sulzberger M. B. and Baer R. L. Year Book of Dermatology and Syphilology Chicago, Y. Bk. Pub. 1949.

IV General Care

- 1 Avoid worry
- 2 Avoid constipation.
- 3 Do not be ashamed to face people because of your skin—your personality will attract people and overshadow your fears.

Apply a little cold cream to upper eyelids and about the mouth so that the lotion will not irritate these parts at night

THE ACNEFORM ERUPTIONS

Occupational Acne

Occupational acne or pseudo-acne (dermatitis industrialis) may result from contact with cutting oils, greases, waxes and chlorine compounds. The characteristic lesions are follicular and pustular with occasional comedones. This type does not appear on those areas involved in ordinary acne vulgaris but affects the thighs, the legs, the abdomen the buttocks, the forearms and the dorsum of the hands and the fingers. Preventive measures include the use of fat solvents, rubber aprons, soaps and hygienic care. Otherwise, the treatment is similar to acne vulgaris except that x-ray therapy is not advisable.

Prognosis. Several months may be required to obtain good results especially if the patient continues to work in the same environment.

Halogen Acne

Iodides and bromides when administered internally often produce a papulopustular eruption on the face, the back, the chest or the legs in susceptible individuals. The lesions are follicular and may bear a close resemblance to those of acne vulgaris. In addition, flare-ups in acne vulgaris often are due to the halogens, although it may be difficult to get a positive history from unintelligent patients. Halogen acne is more inflammatory than ordinary acne and more resistant to treatment unless the underlying cause is discovered. Iodized salt Bromo-Seltzer bromide "nerve tonics" and iodide "blood tonics" are often responsible for this type of acne. The disease is common in epileptics who take bromide medication for long periods of time.

The treatment of this condition is described on page 117

ACNE NECROTICA MILIARIA

Acne necrotica consists of an eruption of pruritic brownish-red papulopustules on the scalp the forehead and the hairline.

The early lesions are blood-crusts and inflammatory the older lesions which undergo central necrosis, tend to form pitted scars, from which the disease acquires its name. Comedones are absent.

Course of the disease is chronic new crops appear from time to time.

Etiology The acne bacillus and the staphylococcus are found in the pustular lesions which are the result of trauma produced by scratching in patients who are under tension.

Prognosis is guarded. Recurrences are common.

Pathology Perifollicular infiltration and necrotic areas are characteristic.

Treatment. Some improvement occurs from the use of 5 per cent resorcinol monoacetate in alcohol during the day and Bac-tracin ointment at night. Phenobarbital or one of the milder anti-histaminics controls the pruritus. If the itching persists and the lesions have healed Quotane ointment is usually effective. Ultra violet radiation is useful in the scalp cases when used in conjunction with antiseptic lotions. Staphylococcus aerobacterin in graduated doses may be effective. Attention to the hygiene of the skin and the scalp and the general health is an important measure in all cases.

ROSACEALIKE TUBERCULID (*Lewandowsky*)

(See p 318)

ROSACEA

This condition is a chronic symptom complex characterized by a symmetrical flushing of the face often associated with acne-form lesions and usually terminating in hypertrophy of the tissues.

Clinical Description. The onset is gradual with temporary blushing. Several months or several years later a permanent flushing (hyperemia) of the face the forehead and the neck appears. This condition, which may persist for a variable period, is followed by the appearance of dilated capillaries, indolent papules papulopustules and dilated follicles in the hyperemic areas. In the course of time the involved skin becomes thickened. After several years the tissues of the nose become hypertrophied in certain individuals and are thrown into bulbous folds (rhinophyma) See Figure 186

ROSACEALIKE KERATITIS occurs in about 5 per cent of all cases of rosacea. The various complications consist of corneal opaci-



FIG. 186 (Left) Rhinophyma as a result of a chronic rosacea

FIG. 187 (Right) Rosacea with infiltration and acneform lesions

ties, ulceration, pericorneal injection and congestion of the palpebral conjunctivae.

Etiology Rosacea appears to be a reflex circulatory disturbance involving the forehead, the nose, the cheeks, and the "V" of the neck, resulting in a persistent dilatation of the superficial and the deep capillaries of the affected parts. These are known as the "flush areas." They are supplied with blood vessels having a delicate vasomotor control. Predisposing factors include seborrhea, chronic gastritis (resulting from rapid eating, caffeine beverages or alcoholism), poor tone of facial muscles, chronic exposure to wind and cold, entropion, infantile uterus, functional utero-ovarian diseases, colitis, intranasal disease (when the rosacea is limited to the nose), high blood pressure, emotional neuroses and hyperthyroidism. The role of the skin saprophyte, *Demodex folliculorum*, is controversial. From a study of the skin temperature in rosacea, Borrie¹ concluded that the arterioles played no part in the pathogenesis of the condition. Over 70 per cent of the cases occur in women from 30 to 45 years of age.

Pathology Section shows dilated capillaries, hypertrophied

¹ Borrie, P. The State of the Blood Vessels of the Face in Rosacea.—J. Brit. J. Derm. 67: 1944.

sebaceous glands and a low-grade inflammatory reaction about the sweat glands and the sebaceous glands.

Diagnosis is based upon the following points (1) occurrence in middle age (2) symmetrical persistent hyperemia (3) limitation to flush areas of face (4) presence of dilated capillaries and acneform lesions (5) absence of scales, scars and atrophy (6) no subjective symptoms except itching in hypertension cases.

Differential Diagnosis

	<i>Rosacea</i>	<i>Acne Vulgaris</i>	<i>Lupus Erythematosus</i>
Primary lesion	Hyperemia	Comedone	Scaly plaque with stippling
Symmetry	Yes	No	Often
Acneform lesions	Yes	Yes	None
Dilated capillaries	Yes	No	Yes
Gastro-intestinal disturbances	Often	Uncommon	None
Atrophy	No	Scars	Yes
Scaling	No	No	Yes
Border of lesion	Ill-defined		Well-defined
Ultraviolet light effect	Worse	Improvement	Flare-up

Treatment. While treating the condition locally an attempt should be made to discover the source of the disorder. The object of treatment should be threefold (1) to abolish the reflexes which finally paralyze the blood vessels of the involved areas (2) to remove the source of local irritation and (3) to remove the results of chronic congestion.

DIETETIC TREATMENT The diet should be bland and should consist of small servings. Alcohol coffee tea and tobacco which arouse the gastric reflex, should be prohibited. Fried, hot and cold foods and iced drinks often aggravate the disease. The patient should eat his meals slowly overeating should be prohibited.

GENERAL THERAPY If gastric analysis reveals a deficiency of hydrochloric acid the condition may be improved by prescribing from 1 to 8 cc. of dilute hydrochloric acid in milk or fruit juice sipped through a glass tube at mealtime. In cases of hyperacidity alkalies (aluminum hydroxide magnesium trisilicate or colloidal kaolin) may reduce the tendency toward flushing. Some investigators minimize the role of gastric acidity in rosacea and emphasize the importance of psychosomatic factors. Constipation should be corrected by the use of nonirritating mineral-oil laxatives. Bile salts, if indicated are useful. In women, small doses of thyroid and estrogens may be useful. If macrocytic anemia is



FIG. 188. (Left) Rhinophyma before electro-surgery (Right) The same case after electro-surgery (From Dr. A. Martin)

present, injections of a liver product or ferrous sulfate capsules t.i.d. are advisable.

PHYSIOTHERAPY X-ray therapy is not advisable except in cases with numerous acneiform lesions which resist local treatment. If no improvement is seen after 350 r. it should be discontinued. Daily facial massage with kneading of the tissues often improves the vascular tone and stimulates the subjacent musculature.

Ultraviolet-light therapy aggravates the disease.

Rhinophyma is corrected with the surgical diathermy loop by shaving off the fibrous tissue with cold steel knife or by dermal abrasion. The dilated capillaries can be destroyed by electrolysis. The negative pole is inserted into the capillary for a few minutes until the vessel turns white.

LOCAL THERAPY Good results often are obtained from local therapy alone. The use of hot water on the face must be avoided. The face should be dried after washing by blotting with a soft towel. Massage, cold creams, strong soaps and "skin foods" are not permitted. Calamine lotion with 3 per cent colloidal sulfur or Kummerfeld's lotion are useful in the hyperemic types. An ointment consisting of colloidal sulfur 4 per cent salicylic acid,

2 per cent, and Quinolol ointment, 5 per cent, in a greaseless base is useful in the acneform types only. This should be discontinued at the first sign of irritation and a soothing cream used (e.g. 5 per cent witch hazel in Aquaphor).

The author issues the following instruction sheet to his patients who suffer from rosacea.

ROSACEA ROUTINE

Please observe the following directions, in order to control your skin condition.

- 1 Do not use hot water on the face.
- 2 Avoid exposure to cold and winds.
- 3 Avoid steam exposure in the kitchen or the laundry.
- 4 Avoid all conditions that cause nervousness and blushing.
- 5 If you have indigestion, avoid the following foods: Fried foods, spicy and hot foods and salty foods like peanuts and popcorn. No carbonated drinks.
- 6 No tea is permitted. Substitute Sanka for coffee. No alcoholic beverages.
- 7 Keep your scalp free of dandruff.
- 8 Avoid constipation.
- 9 Eat slowly and chew food well.
10. Men should use no after-shave lotions.
11. Drink no water WITH meals.
12. Slow down on your activities.

SEBORRHEA

This constitutional condition is characterized by excessive sebaceous activity and a disturbance of fat metabolism. The disease consists of two phases: (1) seborrhea (2) dermatitis. The parts affected are the so-called seborrheic areas, where the glands are most numerous. The most common manifestation of the process is seborrhea of the scalp.

Bacteriology. The *Pityrosporum* of Malassez (a *Monilia*) the acne bacillus and the *Staphylococcus albus* are the triad believed to be responsible for dandruff and seborrheic conditions. A greasy skin is not necessary for the development of these organisms.

Clinical Manifestations. There are two types of seborrhea. The dry type (*pityriasis capitis simplex*) is known commonly as dandruff (see p. 494) and is apparently infectious and autoinoculable. This condition often is associated with premature alopecia which begins on the vertex and frontal regions of the scalp. The oily type (*seborrhea oleosa*) affects both the scalp and the midline of the face and is found more commonly in swarthy individuals. It occurs usually at puberty and may be found about the

ditioris (anemgia) in women. This type often is associated with *acne vulgaris*.

Treatment. The local treatment of seborrhea begins with the proper care of the face and the scalp. In the dry cases, a shampoo every two weeks is indicated. If the hair is fine, and if alopecia is present, a trial of one of the numerous sulfonated soaps is recommended. A brillantine can be applied after the shampoo to restore the luster. For oily scalps, a daily shampoo using plain or the ethereal tincture of green soap is useful. If dandruff is present, a sulfur-salicylic acid ointment in a water absorbent base (p. 529) can be applied the night before the shampoo. A useful alcoholic solution for daily use in mild cases is the modified White lotion

Monooacetate of resorcinol	5 to 10.0
Ethyl alcohol 70% q.s. ad	100.0

8. Apply to scalp at bedtime.

Selenium sulfide (Selsun-Abbott) is also effective and is massaged into the scalp after the usual shampoo permitted to remain on the scalp for 5 to 10 minutes and then is rinsed out. The solution is used every 5 to 10 days depending on the severity of the case. Selsun Cream (Schering) a sodium sulfacetamide product, also may check mild seborrhea.

Ultraviolet radiation in mild erythema doses is stimulating and moderately bactericidal.

SEBORRHEIC DERMATITIS

Seborrheic dermatitis is the second phase of seborrhea, with resulting inflammation (eczema) from bacterial activity in areas characterized by marked pilosebaceous activity. The hypertrophy of the sebaceous glands, the dilatation of the pilosebaceous follicles and the alteration of the composition of the sebum and the fat of the horny layer of the skin favor the growth of the seborrheic triad of organisms.

Clinical Varieties. (1) Lichenoid (2) eczematoid, (3) psoriasisiform (4) follicular (5) pustular and (6) erythematous-squamous.

Clinical Description. The typical eruption usually begins on the scalp and spreads to contiguous areas by extension. After a variable period one or more of the following areas may be involved: eyebrows, eyelids, nasolabial folds, ears, anterior and



FIG. 189 (Left) Seborrheic dermatitis of the scalp
(Right) Seborrheic dermatitis (eczematoid type)

posterior hairlines neck presternal interscapular pubic genital and perianal regions. The scalp type often is followed by permanent alopecia (see p 502). Involvement of the ears, including the shell the retro-aural folds and the external canal is often a diagnostic feature. The eruption on the trunk usually assumes a flower-petal configuration. The flexural type affects the cubital or the popliteal spaces, the submammary folds, the navel or the inguinal folds. This is not uncommon in infants and children. In extensive cases the umbilicus the axillae the submammary and the pubic regions may be affected. Seborrheic cheilitis involves the vermilion border of the upper or the lower lip.

The typical lesions are yellowish-orange oval or round ill-defined patches. They are superficial at the beginning but become more inflammatory and infiltrated at a later stage.

Untreated lesions usually are covered with a dull dirty greasy nonadherent scale or greasy crust. The eruption is not symmetrical. There is slight itching on the nonhairy areas but when the scalp is involved itching may be marked. The disease seldom becomes papular or pustular.

The following diseases may be aggravated by a "seborrheic soil": acne secondary syphilis, occupational dermatitis psoriasis and seborrheic keratoses.

Etiology The exciting cause of the disease is bacterial. The following organisms are often present in the scales *Pityrosporum ovale*, *acne bacillus* and *Staphylococcus epidermidis albus*. In some of the cases there is apparently a seborrheic diathesis or hormonal (stimulation of sebaceous glands) background. Flare-ups may occur from mental strain or from the use of alcohol, greasy foods, pork products and excessive carbohydrates.



FIG. 190 Annular seborrheic dermatitis of face and neck. The lesions are pinkish yellow and are covered with greasy scales.

ciated with greasy skins. Section shows a proliferation of the sebaceous glands. The lesions may be destroyed by light electrodesiccation.

SEBOCYSTOMATOSIS

Sebocystomatosis is characterized by the presence of large groups of pinhead-sized to pea-sized sebaceous cysts. In most of the cases, the scrotum, the trunk and the chest have been involved. The condition is sometimes familial. It occurs chiefly in

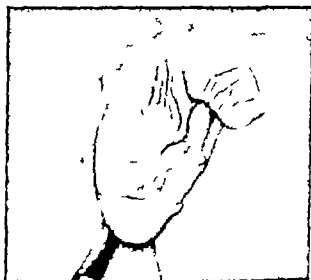


FIG. 191 Multiple sebaceous cysts of the scrotum. Some are calcified, which is not unusual in the aged.

the 20-year-old to 30-year-old age group and probably is a hamartoma.

Treatment consists of excision of the cysts.

NEVIC DISEASES

ADENOMA SEBACEUM

(See p. 591)

SEBORRHEIC KERATOSES

(See p. 377)

NURSING ASPECTS

The patient should be stripped so that the entire sebaceous system may be examined. The weight should be taken and preparations should be made for a complete blood count.

Acne. The patient should be questioned after each ultraviolet light or x-ray treatment regarding any unusual reactions. The same holds true for oral therapy and for injections. Since treatments are usually prolonged, the nurse should give encouragement to the patient and his parents at each visit, so that cooperation and ultimate cure may be obtained. The nurse should inform the physician of any dissatisfaction expressed by the patient or the parent, so that the matter may be investigated, and a change of therapy instituted.

Seborrheic Dermatitis. The nurse should advise the patient regarding the necessity of following the diet, the proper application of local therapy the avoidance of psychosomatic influences, constipation and of strenuous exercise. Sulfa drugs and penicillin ointment must not be used.

Rosacea. The blood pressure, the weight and the basal metabolism, as well as a complete blood count, are needed to evaluate the predisposing factors. Avoidance of the following is important ultraviolet light constipation psychosomatic influences, strong soaps and hot foods and drinks. A typewritten list of "do's and don't's" should be given to the patient.

Diseases of the Sweat Glands

FUNCTIONAL DISEASES

HYPERHIDROSIS

ECRIMIDROSIS

HIDRADENTITIS SUPPURATIVA

NEVIC DISORDERS

SYRINGOCYSTOMA

MALIGNANCIES

INFLAMMATORY DISEASES

SUDAMINA

HYDROCYSTOMA

KERATOLYSIS EXFOLIATIVA

MILIARIA RUBRA

FOX FORDYCE DISEASE

NURSING ASPECTS

Anatomy The sweat apparatus consists of a coil (glomerulus) situated in the deep corium or the hypoderm where the sweat is produced, a spiral duct and a pore. Sweat glands are found everywhere except on the lips, the nail bed and the glans penis. From 700 to 900 cc. of sweat are excreted every 24 hours. The secretion, which is slightly alkaline, has a specific gravity of about 1.004. The glands are supplied with special secretory (adrenergic and cholinergic) fibers from the sympathetic nervous system.

Biologically sweat glands are of two types (1) the small common eccrine glands are distributed generally and are found in largest numbers on the palms and the soles (2) the large sex related apocrine glands, which open into a hair follicle and have a limited distribution in the axillae the pubic region the anogenital area and the breasts. They do not develop until puberty. These glands are involved in Fox Fordyce disease and hidradentitis suppurativa.

The functions of the sweat apparatus include participation in general water metabolism excretion of waste products, regulation of temperature and alkali reserve and control of oxygen-carbon dioxide metabolism of the skin.

The sweat keeps the skin soft and supple. It is also an important factor in the defense of the skin against fungi and bacteria. Its fungicidal action is due to the presence of acetic, propionic, caproic, lactic and other acids. However where the evaporation

of the sweat is retarded as in the axillae the anogenital region and the feet, there is a higher concentration of sugar reducing substances. This state produces a more favorable culture medium for the reproduction of fungi and sets up a pruritus.

When the skin is continuously moistened with sweat, it is more susceptible to contact dermatitis from external irritants than is normal skin. This is an important factor in the predisposition of certain individuals to this condition.

The sweat gland retention syndrome occurs in miliaria (prickly heat) atopic eczema ichthyosis and dyshidrosis as a result of occlusion of the sweat ducts. Sweat retention in the tissues results in pruritus, which is increased in the presence of high temperatures and humidity.

Diseases of the sweat glands consist of functional disorders, disease due to gland obstruction, with or without inflammation, nevic disorders and malignancies.*

FUNCTIONAL DISEASES

HYPERHIDROSIS

General hyperhidrosis, or excess sweating, occurs physiologically from excessive heat, muscular exertion or shock associated with extreme depression (cold sweat).

Pathologically it is found in fevers, wasting diseases, endocrine disorders, viz., acromegaly and exophthalmic goiter and during electric shock or insulin therapy for mental disorders.

Localized hyperhidrosis, a stigma of autonomic imbalance, usually affects the palms, the feet, the axillary regions and the anogenital regions. Due to the anatomy of the involved areas, intertrigo and fungus diseases are common complications. Axillary hyperhidrosis may predispose to contact dermatitis from deodorants, dress shields and dyes in clothing. Excess sweating may occur in acrocyanosis, neurocirculatory syndromes, hyperthyroidism, tachycardia following the taking of certain foods, alcohol, tea and coffee in rare cases it may be an hereditary disorder. Physiologically hyperhidrosis is produced by emotional excitement and tension. The localized type may have a limited distribution along the course of certain nerves following operative procedures.

*For a recent review of the pathology of sweat gland disorders, see Hymowitz, A. B. Some histopathologic aspects of disturbances of sweating. *Arch Dermat & Syph.* 66: 145 1952.

Complications. Axillary sweating predisposes to trichomycosis axillaris, palmar and plantar sweating to dyhidrosis, and excessive sweating of the feet to bromidrosis, monilliasis or dermatophytosis.

Prognosis should be guarded in the neurogenic types.

Treatment. Generalized cases require treatment of the underlying cause. Axillary cases are controlled best with topical applications of a 25 per cent aluminum-chloride solution or any of the commercial deodorants. Contact dermatitis from the use of these preparations is not unusual. When the usual commercial deodorants are not tolerated Allercerme hypo-allergenic liquid deodorant (Texas Pharmacal Co.) may be useful.*

HYPERHIDROSIS OF THE FEET requires special attention to local and general causes, viz., faulty footwear, arch defects, cardiac disease, poor hygiene and emotional instability. Shoes and hose should be changed twice daily. Therapeutic measures include the following:

Solutions: 25 per cent aqueous aluminum chloride 0.1 per cent formalin solution 2 per cent salicylic acid in cologne water

Powder

Boric acid	3
Alum and potassium sulfate	3
Sodium hypochlorite	10
Bentonite	25
Magnesium carbonate q.s. ad	100

S. Put on soles every morning

X-ray therapy may be employed by an experienced dermatologist in those cases where the condition is a serious social or economic handicap.

Anticholinergic Agents. Pro-Banthine (Searle) depresses the postganglionic nerve endings of the parasympathetic system and inhibits hyperhidrosis. The dose is 50 mg. every 6 hours until the condition is under control then a daily maintenance dose of 50 mg. although the dosage must be individualized. In general, results are not satisfactory. Glaucoma and advanced cardiac disease are contraindications.

BROMIDROSIS

Bromidrosis is a state of excessive sweating in which the sweat has a foul odor. The localized type, usually limited to the feet, the axillary spaces or the genitocrural region, results from bac

*For a general review of the subject, see Hermann, F. and Seiberger, M. B. Some aspects of therapy of sweat disturbances, A.M.A. Arch. Dermat. & Syph. 68:162 1952

terial decomposition of the sweat by *Bacillus foetidus* and always is associated with hyperhidrosis. The generalized type may result from the ingestion and the excretion of garlic, sulfur or other aromatic compounds.

Treatment of bromhidrosis consists of frequent hexachlorophene soap-and-water foot baths and the application of from 2 to 5 per cent chromic acid or from 10 to 25 per cent aluminum-chloride solutions daily to the soles of the feet. In addition, the feet should be soaked in a fresh 1:1,000 solution of potassium permanganate twice a day. Shoes and stockings should be changed several times daily if necessary. Patients afflicted with localized hyperhidrosis or bromhidrosis should avoid tennis shoes or rubber soled shoes. Cork soles are very useful in preventing maceration from the footgear. During the summer months patients should be advised to wear perforated shoes and white hose. Barbiturates should be prescribed in cases of emotional instability.

HIDRADENITIS SUPPURATIVA

Hidradenitis suppurativa is a chronic inflammatory condition of the deep apocrine sweat glands of the axillae, the groins and the anogenital regions, consisting of nodules, abscesses, pustules and fistulous sinuses. The primary lesion is a deep-seated furuncle. The disease is characterized by the appearance of red tender nodules which are hard at first and fluctuate later. New lesions occur from time to time. In some cases the disease is aggravated by menstruation.

Differential Diagnosis. Tuberculous adenitis, granuloma inguinale and lymphogranuloma inguinale must be considered. Examination for Donovan bodies should be a routine procedure.

Pathology consists of occlusion and destruction of the apocrine sweat glands, with dissemination of the disease through the lymph channels.

Treatment, in the early cases, consists of Aureomycin therapy (250 mg. t.i.d. for several weeks) and filtered x ray therapy. When the disease is extensive and fails to improve with conservative therapy, total excision, followed by plastic repair is advisable.

NEVIC DISORDERS

SYRINGOMYXOMA

This rare disease consists of an eruption of persistent pale rose papules on the anterior neck, the chest, the breasts and the

abdomen. The disease, which usually makes its appearance at puberty is more common in women. The disorder is considered to be due to imperfect apocrine glands and occasionally to imperfect holocrine glands.

Section shows tubular cordlike epithelial proliferation and numerous dilated cysts lined with epidermal cells morphologically similar to those of the sweat glands and the ducts. The numerous dilated cysts give the corium a "Swiss cheese" appearance. The lesions do not undergo malignant degeneration.

Differential Diagnosis Xanthoma and multiple benign cystic epithelioma may faintly resemble syringocystoma.

Treatment seldom is necessary or desired. Electrodesiccation is usually effective.

MALIGNANCIES

Various types of malignancies may spring from the coil-gland epithelium. In some basal-cell carcinomas, there appears to be an attempt to form sweat glands. Paget's disease (intraepidermal carcinoma) in some cases, may have a definite connection with the apocrine sweat glands. Benign cystic epithelioma may exhibit an adenoid structure from attempts to reproduce the sweat glands.

INFLAMMATORY DISEASES

SUDAMINA

These are minute glassy vesicles appearing on the surface of the skin. They are due to an elevation of the horny layer by an underlying sweat drop. They usually are seen on the trunk in wasting and febrile diseases and are of no significance.

HYDROCYSTOMA

This condition consists of a chronic symmetric eruption of the face characterized by discrete noninflammatory deep-seated clear vesicles of pinhead size to pea size and round or oval in shape. The lesions are translucent and have a central bluish sheen. The disease is limited practically to women who come into occupational contact with steam such as housewives cooks etc. In most cases the eruption is aggravated during the summer months.

Pathology The typical lesions in the corium consist of cystic cavities lined by epithelial cells

Etiology These lesions are retention cysts of the dermal portion of the eccrine sweat ducts. Proliferation of the ductal epithelium also may be a factor in causing the occlusion.

Treatment. The patient should adjust her occupation if possible. Local puncture, followed by astringent lotions, usually is successful. Dermal abrasion may be considered for persistent cases.

MILLARIA RUBRA

(PRICKLY HEAT)

Prickly heat is a sweat-gland retention phenomenon characterized by an eruption of discrete, pinhead-sized papules and papulovesicles having an erythematous halo. The disease is limited to the covered parts of the body involving especially the chest and the back although discrete lesions may extend to the wrists and the ankles. The condition usually occurs suddenly in hot weather following excessive perspiration, humidity poor ventilation high salt intake and nervous tension. Obstruction of the sweat pores by bacteria may be a factor in some cases. Irritation by the clothing and other external factors may render treatment ineffective. In practically all cases there is severe itching and stinging. In consequence, impetigo and furunculosis may be present, especially in children. eczematoid dermatitis and intertrigo are complications in adults.

Cooks, bakers, stokers, engine and boiler room personnel and military-service personnel in the tropics are predisposed.

Pathology The sweat-gland apparatus is normal but the edema of the epidermal cells obstructs the sweat in the ducts. The capillaries and the sweat ducts are dilated.

Differential Diagnosis. Follicular contact dermatitis, follicular early syphilids, dermatophytids and drug eruptions may resemble miliaria.

Treatment. Removal to an air-conditioned room is advisable when possible. Light clothing should be worn to prevent retardation of sweat evaporation. Ointments should not be used because of the acute inflammatory character of the disorder. In cases associated with intolerable itching, colloidal baths should be taken morning and night. Local applications should be restricted to bland powders since the condition is self limited if the provocative

abdomen. The disease, which usually makes its appearance at puberty is more common in women. The disorder is considered to be due to imperfect apocrine glands and occasionally to imperfect holocrine glands.

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factors are eliminated. Irritating topical applications may cause incapacitation.*

The following powder may be applied to the affected parts

Camphor	0.5
Pulverized boric acid	2.0
Salicylic acid	0.5
Powdered talcum q.s. ad	100.0

Internally the use of alkaline citrate salts is of value.

KERATOLYSIS EXFOLIATIVA

This benign condition consists of very superficial tiny dry thin scales usually limited to the palms, absence of inflammation and true vesicles. It is symptomless and often is recurrent.

Etiology It is seasonal usually appearing in warm weather. The mosaic fungus has been found in some cases. Hyperhidrosis is common.

Treatment. Although the disease is self limited, a 3 per cent solution of salicylic acid in alcohol often hastens resolution.

FOX FORDYCE DISEASE

This condition is characterized by an eruption of extremely pruritic papules involving the axillae and the nipples, the pubic and the perineal regions. The eruption consists of indolent firm, pale papules with intervening pigmentation. In some cases the skin is slightly edematous.

Etiology Two theories are held (1) apocrine gland sweat retention with pore occlusion and (2) a disturbance of the gonadotropic hormones. The endocrine theory is strengthened by the fact that the disease often clears up during pregnancy†

Section shows a marked dilatation of the apocrine glands, with degenerative changes in the lining epithelium and inflammation about the related hair follicles.

Treatment consists of mild antipruritic lotions and estrogenic substances, which are of value occasionally. If x ray therapy is ineffective plastic surgery should be considered.

*See Shelley W. B. and Simons, R. D. G. The miliaria group of dermatoses, chap. 32 in Simons, R. D. G. editor: Handbook of Tropical Dermatology Houston, Elsevier 1954.

†For a review of the subject, see Cornbleet, T. Pregnancy and apocrine gland diseases: Hidradenitis and Fox Fordyce disease, A.M.A. Arch. Dermat. & Syph. 65: 12 1952.

NURSING ASPECTS

The patient complaining of disease of the sweat glands should be examined in a warm room so that chilling may be avoided and the sweat may be observed under the most favorable conditions. The temperature, the pulse rate and the presence or the absence of a foul odor should be recorded. General hyperhidrosis is more important as a symptom than the localized variety since it may indicate tuberculosis, diabetes or exophthalmic goiter.

Localized Hyperhidrosis. This condition may predispose to dyo dermatitis, fungus infections and impetigo. The nurse should advise the patient regarding frequent changes of underclothing, bath hygiene, the use of powders during the day and the use of astringents at night, and the influence of psychosomatic stimulation on the condition. The possible influence of occupation should be investigated. Hyperhidrosis in infants may predispose to furuncles, miliaria (prickly heat) or impetigo.

Miliaria. This condition may be a problem in industry. Proper ventilation, antihumidity measures, the avoidance of alcohol and of hot drinks, frequent changes of porous clothing and rest periods to avoid overexertion are important prophylactic suggestions. The avoidance of "home remedies," the relief of pressure from clothing to avoid eczematization and the application of antipruritic lotions rather than ointments are important in the management of this minor but uncomfortable dermatosis.

Diseases of the Nails

DISEASES PECULIAR TO THE NAILS	NAIL MANIFESTATIONS OF
ACUTE PARONYCHIA	DERMATOSES (<i>Conid</i>)
ONYCHOMYCOSIS	ACUTE AND SUBACUTE
FAVUS	LUPUS ERYTHEMATOSUS
INGROWING TOENAIL	OCCUPATIONAL DISEASES
HANGNAILS	NAIL MANIFESTATIONS OF
HEMATOMA	SYSTEMIC DISEASES
NEW GROWTHS	SYPHILIS
ONYCHOMADESIS	TUBERCULOSIS
LEUKONYCHIA	TRICHINOSIS
ONYCHORRHIXIS	LEPROSY
BEAU'S LINES	ARTHRITIS
PIGMENTATION	ENDOCRINE DISORDERS
ONYCHOPHAGIA	HYPOVITAMINOSIS
HYPOCRATIC NAILS	NUTRITIONAL ANEMIA
FRAGILITAS UNGUIUM	PLUMMER VINSON
ONYCHAUXIS	SYNDROME
ONYCHATROPHIA	FRACTURES
SUBUNGUAL HYPERKERATOSIS	CERVICAL RIBS
KOILONYCHIA	CONGENITAL DISEASES OF THE
ONYCHOLYSIS	NAILS
NAIL MANIFESTATIONS OF	CONGENITAL ECTODERMAL
DERMATOSES	DEFECT
CONTACT DERMATITIS	PACHYONYCHIA CONGENITA
DERMATITIS EXFOLIATIVA	GENERAL DIAGNOSIS OF NAIL
ALOPECIA AREATA	DISEASES
ICHTHYOSIS	NURSING ASPECTS
EPIDERMOLYSIS BULLOSA	

Anatomy The nails are cutaneous appendages designed to protect the extremities of the fingers and the toes from external injury. Each nail consists of plate, lunula, matrix, bed, nail fold and cuticle. The nail bed upon which the nail plate rests,

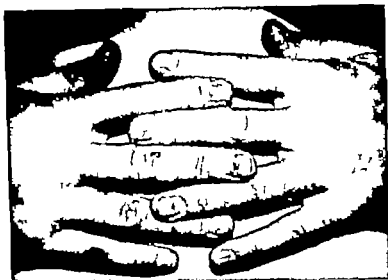


FIG. 192 Dystrophy of the nails in a case of chronic cutting-oil dermatitis.

consists of vascular sinuses, rich nerve trunks and fibers and vascular end-organs. These vascular structures, the glomus bodies, are said to regulate the blood pressure. The average rate of growth in a month is $\frac{1}{8}$ in. (Sequeira) in health Bean* did not observe a seasonal variation in growth in a careful study

THE NAIL SUBSTANCE itself is composed chiefly of keratin and cystine but the natural lipids are mostly cholesterol.

Nail disorders consist of a variety of congenital and acquired diseases, including nevi dystrophies, inflammations, injuries, tumors and infections. Since nail involvement occurs in about 20 per cent of all cases of ringworm of the fingers and the toes, onychomycosis ranks next to trauma and pyogenic infection as a cause of nail diseases †

The various pathologic disturbances of the nails interfere with their smoothness texture color and growth

*Bean, W. B. A note on fingernail growth, *J. Invest. Dermat.* 20:27 1953

†For a detailed discussion of the subject, consult Pardo-Castello, V., *Diseases of the Nails*, Springfield, Ill. Thomas, 1941

Classification is incomplete but the following outline is useful

- 1 Congenital disorders
- 2 Traumatic conditions
- 3 Conditions caused by chemical or physical irritants
- 4 Occupational diseases
- 5 Pyogenic infections with secondary changes in the nails
- 6 Fungous diseases
- 7 Psychosomatic conditions including nail biting
- 8 Disorders of growth due to systemic diseases (psychiatric, circulatory nutritional and endocrine)
- 9 Conditions associated with dermatoses (psoriasis, exfoliative dermatitis, alopecia areata, etc.)
- 10 Drug eruptions (usually pigmentations)
- 11 Nail disorders due to nerve lesions
- 12 New growths
- 13 Unknown causes

DISEASES PECULIAR TO THE NAILS

ACUTE PARONYCHIA

Paronychia is an acute infection of the nail fold due to various causes and is characterized by redness, swelling and tenderness. The pain is due to the inexpandibility of the area and the nerve supply to the nail bed.

Etiology Acute paronychia is often secondary to trauma, hangnails, nail biting dermatitis industrialis, scabies impetigo, etc. It is common in "pickers" as a result of nervous tension. In the common pyogenic type the *Staphylococcus pyogenes* usually is found.

Prophylaxis. The hands must be kept out of water. No dish-washing should be permitted.

Differential Diagnosis. The pyogenic types are more acute than the mycotic forms, which usually affect more than one nail. A drop of pus expressed from the lesion should be examined for pyogenic organisms.

Treatment consists of the application of hot antiseptic wet packs to hasten suppuration. When pointing occurs, a linear incision should be made parallel to the nail fold, and a small iodo-form drain should be inserted to favor drainage.

Chronic Paronychia

Chronic paronychia, which is mainly due to *Monilia albicans* and rarely to other yeasts and fungi is common in housewives and dishwashers. Frequent immersion of the fingers in dish water favors infection. Fruit canners are predisposed. Extension of the process may result in involvement of the nail root. If microscopic examinations of the pus or the scrapings are negative, routine cultures should be made.

Treatment. Useful procedures are (1) the daily local application of a 2 per cent alcoholic solution of gentian violet, (2) a protective dressing, (3) the avoidance of dishwashing, (4) avoidance of manicures and (5) antibiotic or Vioform creams.

Resistant cases are treated best by vaccines or x ray irradiation to the affected part, with protection to the nail root.

Onychomycosis

Onychomycosis (ringworm or *tinca unguis*) comprises about 20 per cent of all nail diseases. The infection usually begins as a cloudy patch on the lateral surface of the nail and extends gradually toward the nail root. The distal end becomes thinned out, leaving a reedy frame but the entire nail rarely is destroyed. When fungi are the causative factor the nail plate appears to be worm-eaten or squamous and is brownish or black in color. In other cases there are desquamation and longitudinal striations. *T. purpureum* infections should be suspected when the disease is resistant to treatment. These infections are characterized by deep involvement. *T. gypsum* infections are characterized by superficial involvement and monilia infections by paronychial involvement.

Etiology. The disease is caused by a heterogeneous group of organisms, including dermatophytes, achorions, monilia and rarely saprophytes. Yeasts not infrequently cause chronic paronychia.

Prognosis. The disease is notoriously stubborn to treatment. A year or longer may be required for satisfactory results.

Diagnosis. Microscopic examination and attempts to grow the causative organisms on culture media are not always successful. The nail shavings should be soaked for 14 hours in 20 per cent potassium hydroxide and then should be examined with the high-power lens.

Differential diagnosis is mainly from psoriasis when it is limited to the nails. This condition is more likely to affect the nails of the fingers, rather than of the toes. Involvement is symmetrical. Subungual hyperkeratosis are more marked. Punctiform depressions are present and microscopic and cultural studies are negative for pathogenic fungi or yeasts.

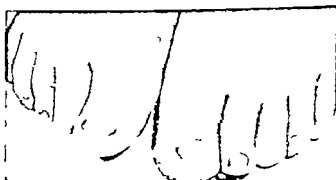
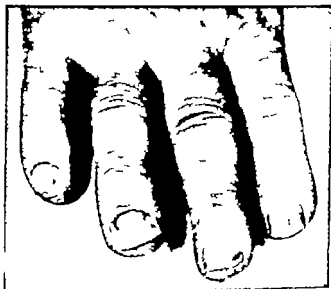


FIG. 193. (T. P.) *Onychomycosis*. The nail plate is destroyed eventually by the fungi. (Bottom) *Onychomycosis* (*T. gyrans*)

Treatment is unsatisfactory. First, it is necessary to discard the infected shoes and stockings. As much of the infected nail plate as possible should be removed by applying potassium hydroxide and then grinding with the electric drill. At night, it is advisable to apply full strength Whitfield's ointment (compound benzoic-acid ointment). Ammoniacal silver nitrate solution is rarely effective. Thorium γ ointment may be beneficial. In recurrent cases, x ray therapy is effective occasionally but should be used with caution. Avulsion of the nail may result in complete eradication of the disease if fungicidal therapy is continued during the period of regrowth, although Cleveland White advises against surgical measures because of the frequency of recurrences in the new nail. Lithium bromide 20 per cent in linoleum lacquer (Dupont) or Verdefam (Texas Pharmacal Co.) applied daily for several months may prove satisfactory in some cases.

FAVUS

In rare instances the nails may be infected with the *Achorion schoenleinii* resulting in a yellow friable haterless hyperkeratotic or honeycombed nail plate. A greenish fluorescence is present under the Wood light in untreated cases. Latent or active lesions will usually be found in the scalp. (See p. 490.)

INGROWING TOENAIL

Ingrowing toenail (onychocryptosis) is an acute inflammatory condition of the soft tissues at the corner of a toenail. The nails of the great toes are involved commonly.

Etiology includes (1) faulty foot balance which throws a distorting stress on the great toe, (2) short pointed shoes which make pressure on the nail of the great toe, (3) careless and improper trimming of the nails, (4) excessive width and abnormal convexity of the nail.

Symptoms include pain, tenderness, swelling and redness at the corner and along the lateral margin of the nail. Frequently infection of the involved area with pus formation, occurs. After several days the deep tissues become necrotic, and a moist area covered with sluggish granulations develops.

Prophylaxis includes wearing the proper type of shoe and correcting faulty foot balance.

Treatment. If infection and redundant granulations are present cotton packing soaked in a 10 per cent solution of silver

nitrate is inserted into the nail fold. This usually destroys the excess granulations and clears up the infection.

Severe cases of "ingrowing toenail" require operative procedures the details of which can be found in general works on surgery.

HANGNAILS

Hangnails are tags of the nail fold due to various causes, including xeroderma, strong acids or alkalis, poor hygiene, careless manicuring or nail biting. Since they may become a portal of infection to pathogenic organisms, they should not be neglected.

Treatment, which is simple, consists of trimming them closely with a sterile scissors and sealing the base of the lesion with flexible collodion.

HEMATOMA

Hematoma (hemorrhage) may be caused by trauma, sulfa drugs, blood dyscrasias or vitamin C deficiency. The nail usually appears discolored from the pool of blood beneath it. In severe cases a hole may be drilled in the nail plate over the center of the lesion to relieve the tension.

NEW GROWTHS

New growths involving the nail region are uncommon. The benign growths include verruca vulgaris, granuloma pyogenicum, pigmented nevus, chondroma, angioma, periungual fibromata (associated with adenoma sebaceum), clavus and glomus tumors.

The malignant types are rare. Occasionally melanoma develops in the nail bed. Squamous-cell carcinoma is rare and may arise secondary to arsenical keratoses.

ONYCHOMADESIS

Onychomadesis consists of a separation of the nail from the bed starting at the base. After the loss of the old nail, replacement takes place within six months. This shedding may follow exfoliative dermatitis, trauma and various infectious fevers.

LEUKONYCHIA

Leukonychia is a common condition characterized by the appearance of white spots or striae in the nail plate. In rare cases, the entire nail plate is affected (leukonychia totalis).

Etiology. In most cases the cause is unknown. If unilateral the cause may be a circulatory disturbance. Other causes are emo-

tional upsets, trauma, and the presence of a severe systemic infection. The condition may be due to a persistence of the keratohyalin granules, following increased metabolic changes in the nail. Treatment is unsatisfactory.

ONYCHORRHEXIS

Onychorrhexis is a condition characterized by longitudinal striations, with or without fissures. The free edges of the nails are thinned and break off or split easily.

Etiology includes trauma, senility, angiospasm, hypocalcemia, extreme dryness from strong alkalies, degreasing agents (acetone, carbon tetrachloride, benzene), nail polish removers and diseases of the nervous system.

Treatment consists of the daily application of silicone protective ointments and large doses of vitamin A (50,000 units daily for at least 3 months). Amino acids in the form of gelatin should be added to the diet. In persistent cases, a trial with thyroid extract over a period of several months may be rewarding.

BEAU'S LINES

Beau's lines are transverse lines, or superficial sulci, which give the nail a wavy appearance. They are due to a sudden arrest of function of the matrix, with disturbance of normal nail production (keratinization). They occur in acute infectious diseases, early syphilis, tuberculosis and nervous conditions.

PIGMENTATION

Pigmentation of the nail bed may occur in argyria and Addison's disease. Phenolphthalein, ACTH and cortisone and quina crine may produce a similar condition. Exposure to radiation, resorcinol and base-cure reactions may be followed by discoloration of the nails.

ONYCHOPHAGIA

Onychophagia (nail biting) is associated with chronic paronychia and hangnails in unstable or tense individuals. The condition is an infantile habit or an expression of suppressed aggression.

Treatment is unsatisfactory until the psychosomatic cause is found and controlled. In some cases, the application of quinine ointment may discourage the habit.

HIPPOCRATIC NAILS

Hippocratic nails often are associated with club fingers, which usually are diagnostic of chronic cardiac or pulmonary disease.

FRAGILITAS UNGUIUM

Fragilitas unguum (brittle or eggshell nails) usually results from too-frequent manicuring or the use of alkaline cuticle softeners or enamels.

ONYCHAUXIS

In onychauxis (hypertrophy), the nail is thickened distorted and enlarged to an extreme degree. The nail plate may increase greatly in size in the aged and in various trophic disturbances, so that it resembles a thick horny plate. The condition also occurs in mental disease, chronic dermatoses and acromegaly. When the hypertrophy assumes a ram's-horn appearance, it is known as onychogryphosis.

Treatment consists of avulsion in the extreme cases, although most patients seem to tolerate the condition as a pet deformity.

ONYCHATROPHIA

Onychatrophia is characterized by the presence of small nails or thin stumps. The condition is due to various causes, including occupation, infection, trauma, systemic disturbances and endocrine disturbances.

SUBUNGUAL HYPERKERATOSES

Subungual hyperkeratoses consist of growths of heaped-up hyperkeratotic epidermis under the free edge of the nail. They are due to occupation, base-coat manicure preparations, fungus infection, psoriasis and other chronic dermatoses. It is important to make sure that the lesion is not a verruca.

Treatment consists of curettement of the lesion and the application of a 4 to 10 per cent salicylic-acid ointment.

KOILONYCHIA

Koilonychia (spoon nail) is a manifestation of a disturbance of the iron metabolism in which the nail assumes a concave surface. The condition is a part of the Plummer-Vinson syndrome. (See p. 571.) Treatment consists of therapy with iron, riboflavin or liver.

ONYCHOLYSIS

Onycholysis is a term used to describe the separation of the nail from its bed by a curvature of the nail plate.

Etiology Chronic trauma prolonged immersion in alkaline soap solutions, hypothyroidism arthritis and subungual growths may be factors.

Treatment consists of giving vitamin D₂ for several weeks.

NAIL MANIFESTATIONS OF DERMATOSES

PRURIARIS

(See p 127)

CONTACT DERMATITIS

Nail changes occurring in this condition are usually secondary to involvement of the nail fold and the matrix. These cases are complicated often by secondary infection with the staphylococcus. The nail plate may become detached, but regrowth usually occurs.

Nail polish, cuticle softeners and removers may produce eruptions in those who are hypersensitive to them. The dermatitis not only may be limited to the periungual area but also may involve the eyelids the face and the neck.

DERMATITIS EXFOLIATIVA

Nail involvement occurs early in this disease. The nails first become brittle and later are shed. Regrowth is slow and results in irregular nails.

ALOPECIA AREATA

Nail changes when they occur in this disease consist of Beau's lines, brittleness and other trophic disorders.

ICHTHYOSIS

Subungual hyperkeratoses are often found in this condition.

EPIDERMOLYSIS BULLOSA

Loss of the nails, with scar tissue replacement is a common complication in the dystrophic types. The nail changes are sometimes a diagnostic feature.

ACUTE AND SUBACUTE LUPUS ERYTHEMATOSUS

Pink vascular areas are sometimes present in the nail beds and the nail folds.

OCCUPATIONAL DISEASES

Infections, mechanical and chemical irritation as well as pigmentation of the nails may occur in industry. Some of these nail infections may become incapacitating.*

NAIL MANIFESTATIONS OF SYSTEMIC DISEASES

Examination of the nails may reveal the presence of chronic endocrine visceral psychosomatic or toxic conditions.†

SYPHILIS

Chancres of the fingers which are rare usually involve the nail fold. They often are painful and are mistaken for paronychia. Resistance to local treatment, refractoriness following incision, and involvement of the epitrochlear glands should awaken suspicion. The index finger is frequently involved.

Early Syphilis. Shedding of the nails may occur with or without an associated syphilitic alopecia.

Gummas of the nail region are rare.

Congenital syphilitic changes in the nails are not distinctive. However the triad of dactylitis of the terminal phalanx, paronychia and nail changes is pathognomonic.

TUBERCULOSIS

Transverse ridging and pitting of the nail plate and leukonychia are common findings in chronic pulmonary tuberculosis. In tuberculous paronychia the nail bed is dusky red and edematous.

TRICHINOSIS

Splinter hemorrhages beneath the nails are not uncommon. They probably are caused by plugging of the capillaries.

LEPROSY

Leprosy in both cutaneous and nerve types is characterized by

*For a complete discussion of the subject, see Schwartz, L., Tailpan L. and Peck, S. M. *Occupational Diseases of the Skin*, chap. 44 Philadelphia, Lea & Febiger 1947.

†Berson, E. S. Nail examination as a diagnostic aid, *J. Amer. Med.* 61: 38, 1950.

various changes in the nails which are often of diagnostic importance. Complete loss of the nails, as well as atrophic changes in the bones, are common findings.

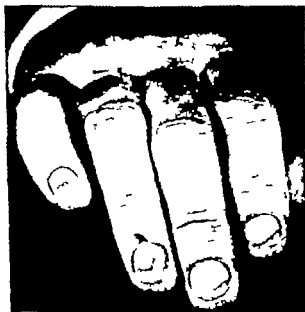


FIG. 194 Syphilitic oostydia with fractures of the nail plate from slight trauma in a case of secondary syphilis. Regrowth followed specific therapy.

ARTHRITIS

The nails may exhibit various forms of dystrophies in chronic arthritis. Ridging occurs in the acute types.

ENDOCRINE DISORDERS

In myxedema and hypoparathyroidism the nails are usually thin, brittle and dry. Hypertrophic changes are common in acromegaly.

HYPOVITAMINOSIS

Vitamin deficiency rarely causes nail disturbances. Shedding of the nails may occur in cases of pellagra.

NUTRITIONAL ANEMIA

Secondary anemia may result in koilonychia, brittleness, convex nails or exfoliation of the free edges.

PLUMMER VINSON SYNDROME

Plummer Vinson syndrome is characterized by the occurrence of spoon nails, secondary anemia, cheilitis and dysphagia. (See p. 571)

FRACTURES

Beau's lines frequently occur following fractures, as a result of nutritive disturbances in the affected extremity

CERVICAL RIBS

Cervical ribs indirectly may cause onychodystrophies. In some cases there are atrophy of the terminal phalanx and neuritic pains in the upper extremities.

CONGENITAL DISEASES OF THE NAILS

CONGENITAL ECTODERMAL DEFECT

In this condition the nails are usually friable thin and brittle.

PACHYONYCHIA CONGENITA

This is a rare nevold condition and is characterized by thickening or complete atrophy of the nails, with subungual palmar or plantar hyperkeratoses. Usually it is associated with keratosis pilaris, cheilosis, extensive nevi of the skin and the mucous membranes.

GENERAL DIAGNOSIS OF NAIL DISEASES

1 The diagnosis of most nail disorders is difficult even when observed by experienced clinicians.

2 Widely different causes may produce the same clinical pattern, and the same cause may result in different nail features.

3 The difficulty of making biopsies in nail disorders is one reason for gaps in our knowledge of these conditions.*

*White, C. J. and Lappay T. C. Histopathology of nail diseases, J Invest. Dermat 19 121 124 1952

4 Nail diseases in the senile are usually caused by circulatory disorders and are resistant to therapy

5 Nail disturbances associated with dermatoses are the easiest to diagnose.

6 Rule out heredity by duration of disease and presence of other anomalies.

7 Rule out systemic disease by history and physical examination. Involvement tends to be symmetrical but this is not always true. Examine for syphilis, tuberculosis, vitamin deficiencies, anemia, cardiac diseases, nervous disorders, and especially endocrine conditions.

8 Rule out occupational disease by history

9 Rule out local causes. When limited to one nail trauma or a recent paronychia may be the cause. Look for evidence of dermatophytosis. Examine entire body for psoriasis. Inquire about manicure preparations and washing powders and soaps.

10 Examine deep nail scrapings for fungi if onychomycosis is suspected.

11 Ecchymoses may be a manifestation of drug sensitivity

12 Unilateral disturbances of growth may be of circulatory or neurogenic origin.

13 Calcium deficiency rarely causes nail disturbances in spite of the popularity of this theory

14 New growths in the nail area should be biopsied to rule out malignancies.

NURSING ASPECTS

Even if the condition is limited to the toenails, the fingernails must be examined also, and vice versa. The nurse should have available the following items: instruments for removing part of the nail for cultural purposes; a nail drill; slants of Sabouraud's agar; clean glass slides; 5 per cent silver nitrate solution; tincture of iodine; 1 per cent iodine in xylene; 5 per cent chrysarobin in chloroform; and a biopsy set for tumors of the nail. If a systemic cause is suspected, a routine Kahn test, a basal-metabolism test and a complete blood count may be ordered by the physician. In any case the patient should be stripped, and the entire integument should be examined for evidence of psoriasis, ichthyosis, chronic stasis eczema, etc.

Diseases of the Mucous Membranes

DISEASES OF THE LIPS

CONTACT CHEILITIS
CHEILITIS EXFOLIATIVA
RETENTION CYSTE
CHRONIC MACROCHEILIA
KERATOSES
VARICOSITIES
OTHER CONDITIONS

DISEASES OF THE MOUTH

LEUKOPLAKIA
FORDYCE'S DISEASE
STOMATITIS VENENATA
BERKET'S SYNDROME
APHTHOUS STOMATITIS
ORAL MONILIASIS
VINCENT'S INFECTION

DISEASES OF THE TONGUE

VERRUCA
CHANCER OF THE TONGUE
TUBERCULOSIS

DISEASES OF THE

TONGUE (*Contd*)

BLACK Hairy TONGUE
PURPURA OF THE TONGUE
SCROTAL TONGUE
PAPILLITIS
BURNING TONGUE
GEOGRAPHIC TONGUE
MOELLER'S GLOSSITIS
GLOSSITIS RHOMBICA MEDIANA
SMOOTH ATROPHY OF THE
TONGUE

MISCELLANEOUS CONDITIONS

OCCUPATIONAL CONDITIONS
ORAL MANIFESTATIONS OF
SYSTEMIC DISEASES

MUCOUS MEMBRANE

NEUROSIS

PLUMMER-VINEON SYNDROME

NURSING ASPECTS

The mucous membranes are subject to various conditions but, due to secondary maceration and the activity of the normal bacterial flora they all tend to assume a similarity in appearance.

Mouth lesions may be independent conditions (leukoplakia) manifestations of systemic diseases (pellagra syphilis, etc.) or mucous-membrane complications of dermatoses (erythema multiforme lichen planus or lupus erythematosus)

Genital Mucous Membranes. The normal vaginal secretions being acid the vagina is usually free of bacteria as a result of their bactericidal action. The urethral meatus in both male and

female are also sterile resulting from the flushing action of the slightly acid urine.*

Flora of the Mouth. The normal flora varies according to age, habits, season, mouth hygiene and the general health. Organisms present at one time or another include streptococci staphylococci, micrococci, lactobacilli, gram-negative coliform bacilli, *T bac cells* *T microdentium* and *macrodentium* diphtheroids, *C albi* *cous* fusiform bacilli, *Borrelia vincentii* and aerobic actinomyces. The growth of these organisms is restrained by the flushing action of the saliva which is mildly bacteriostatic.

The diagnosis of mucous-membrane lesions from a bacteriologic standpoint, is made difficult by the fact that various yeasts, spirochetes and bacteria are part of the normal flora and their significance, when associated with lesions, must be taken *cum grano salis*. The physician should be familiar with the normal appearance of the oral and vulvar mucous membranes the gingival tissues and teeth at the various age levels.

Biopsies are necessary in all lesions of the mucosa where the diagnosis is not clearly evident, when carcinoma is suspected and when the lesion does not respond to indicated therapy †

Complete blood counts should be made in persistent, resistant and necrotic infections, cases of recurrent bleeding a history of bleeding or the presence of petechiae persistent glossitis and atrophy of the lingual papillae.

Incidence. F P and P L. McCarthy‡ list the following conditions as most frequently occurring in office practice chronic gingivitis, chronic aphthous stomatitis, papillitis, chronic cheilitis, squamous carcinoma of the lip fibrous epulis, hemangioma and mucous cysts of the lip.§

DISEASES OF THE LIPS

CONTACT CHEILITIS

Contact cheilitis is an acute a subacute or a chronic inflammation of the lips due to various local causes. It is characterized

Burrows, W. Textbook of Bacteriology p. 227 Philadelphia, Saunders, 1950.

†For the proper technic, see Steepack, D. C. Biopsy technique in oral surgery Oral Surg., Oral Med. & Oral Path. 5:1034, 1952.

‡McCarthy F. P. and McCarthy P. L. Disease of the mouth. A statistical review of 4,778 patients, New England J. Med. 250:493, 1954.

§For a more detailed study of oral diseases, see Barkert, L. W. Oral Medicine, ed. 2, Philadelphia, Lippincott, 1952 and Miller S. C., Oral Diagnosis and Treatment, ed. 2 New York, Blakiston, 1946.

DISEASES OF THE MOUTH

LEUKOPLAKIA

Leukoplakia ("smoker's patches") is a chronic inflammation of the mucous membrane of the mouth especially the buccal mucosa, characterized by the gradual development of irregular milk white dry patches of keratinized epithelium with a tendency to malignant degeneration. The vulvar and the anal mucosae may be affected also. The condition may be limited to the gums in edentulous patients.

Clinical Description. The onset is gradual with the formation of small reddish areas which gradually assume a white surface. By coalescence, the lesions form a patch which spreads slowly in an irregular manner. As it becomes more and more infiltrated, the surface becomes rough, rugose, desquamating or fissured.

Three varieties are recognized (1) smooth (2) raised-plaque and (3) verrucous types depending upon the duration, the hygiene of the mouth and intrinsic factors. In color the lesions vary from a white-enamel to a grayish-blue or a dirty yellow color.

The disease is usually symptomless, although there is some hypersensitivity to heat and cold as well as a mawkish taste, common to all chronic tobacco-users.

Etiology. Leukoplakia is a disease of past middle life consisting of an inherent tendency to hyperkeratosis of the mucous membranes following irritation. Most of the cases are seen in the 50-year-old to 60-year-old age group. It is not extraordinary that 95 per cent of the cases occur in men, who are the heavier smokers and who pay less attention to dental hygiene. For some unknown reason, the disease is rare in the Negro.

Tobacco is the chief predisposing cause. The irritant factor is not the nicotine but the hot smoke, the phenols, the tars and the pyridine content. In the order of importance as irritants are pipes, cigars and cigarettes.

POOR ORAL HYGIENE is second to tobacco in importance. Poorly fitting vulcanized dentures and bridges, snags, malposed and misshaped teeth may be important sources of irritation.

ALCOHOL if used excessively by tobacco users, apparently intensifies the irritating effects of the latter.

Pathology. Keratinization is a defense mechanism in which the normal transitional epithelium is replaced by the stratified

squamous type of cell. In simple leukoplakia, hyperkeratosis, granulosa, acanthosis, with distortion of the rete pegs, and a slight cellular infiltration in the upper corium are typical. As the result of persistent irritation the hypertrophied rete pegs may penetrate through the basal membrane, with atypical cell proliferation and resulting malignancy.

Prognosis. When the disease is recognized early the use of tobacco is discontinued, and proper attention is given to mouth hygiene, slow spontaneous involution is the rule. Malignancy usually follows the verrucous type. Unskillful treatment stimulates all types.

Differential Diagnosis. Leukoplakia must be differentiated from scars, lichen planus and mucous patches. Leukoplakia is rare in women, is apt to be localized over an area subject to irritation by a jagged tooth or rough dental appliance, does not take up iodine tincture unlike lichen planus and does not have the annular or star like pattern. In lichen planus there are usually satellite lesions in the vicinity of the plaque, as well as cutaneous lesions elsewhere. Leukoplakia fluoresces under the Wood light (vivid bluish white) while lichen planus does not. Mucous patches are more inflammatory, have a more friable surface and a shorter duration. In doubtful cases a biopsy and serologic tests settle the question.

Treatment. Before treating a case of leukoplakia it is important to recall the phrase *Primum non nocere*. More patients with leukoplakia have been harmed than benefited by unskillful or energetic treatment. Since carcinoma develops in from 15 to 20 per cent of the advanced cases, any procedure tending to irritate the lesions is decidedly dangerous.

If attention is paid to the following rules, the great majority of early cases of leukoplakia will involute spontaneously.

1. Forbid the use of tobacco and alcohol.
2. Do not use caustics or the silver-nitrate stick.
3. Adjust all artificial dentures and poorly fitting bridges.
4. Remove tartar and trim off all rough and jagged teeth.
5. Treat and correct all orthodontic anomalies.
6. Treat syphilis, if it is present, with penicillin.
7. If cancerphobia is present counteract it by discussion.
8. Early lesions may respond to vigorous scrubbing daily with Ivory soap on a tooth brush.

RADICAL TREATMENT which is seldom necessary consists of excision or surgical diathermy. In the ulcerated and the fissured types, cautery destruction is a useful procedure. If the lesion appears to have undergone malignant degeneration a biopsy should be made at once and serial sections should be examined by a competent pathologist. If carcinoma is present, surgical consultation is necessary.

FORDYCE'S DISEASE

Fordyce's disease is a common condition of the mucosa of the cheeks, the lips or the gums and is characterized by superficial pinhead-sized white or yellow elevations. They are most numerous along the line of the teeth. In most cases they do not appear until puberty after which they persist indefinitely. The lesions have been found also on the glans penis and the labia minora. About 70 per cent of all adults are affected.

The condition is not a disease but is a sebaceous nevus, consisting of ectopic, misplaced or embryologic sebaceous glands. Since the lesions are benign and symptomless, no treatment is necessary but cancerphobia should be counteracted by discussion.

STOMATITIS VENENATA

This condition is caused by repeated contact of various chemical substances on sensitized tissues in the mouth.

Clinical Data. The clinical lesions range from erythema to superficial ulceration depending upon the cause, the duration of contact and secondary factors. The symptoms are out of proportion to the severity of the stomatitis.

Diagnosis is made by careful history and patch tests.

Etiology. The following substances may be causative: dentifrices, antibiotic products, mouthwashes and dental applications, metallic, vulcanite and acrylic dentures, metallic fillings, and denture creams or powders.

Treatment consists of avoiding the cause and prescribing local therapy as indicated.

BEHCET'S SYNDROME

This is a chronic recurrent condition in young persons characterized by aphthous ulcers in the mouth, chancreoidlike ulcers of the genitalia and eye involvement. In some cases, the central nervous system may be affected (Curth). The three signs may not occur

simultaneously Erythema-nodosum lesions may appear in some cases. The cause is not known, but a systemic virus has been suspected. Treatment is symptomatic. Chlorotetracycline or chloramphenicol may be effective but if results are not obtained after one week, ACTH, cortisone or intravenous triple typhoid injections are advisable if there are no contraindications.

APHTHOUS STOMATITIS

Herpetic stomatitis consists of an eruption of one or more yellowish superficial vesicles which, upon rupture become shallow grayish erosions surrounded by an erythematous halo. The acute type clears up within a week, but the chronic recurrent form may persist for several months.

The lesions may be tender and painful. In some cases they are discovered accidentally. As a rule, the ulcers appear on the edge of the tongue or on the buccal mucosa. Recurrences are the rule. In infants, the condition may be associated with mild fever and gastro-enteritis.

Etiology The condition is due to a virus, although persistent cases may be associated with avitaminosis B₁, psychosomatic or bacterial influences. Acute cases may be caused by allergy to specific foods (e.g. nuts) or by gastro-intestinal upsets.

Prognosis. Mild cases clear up within a week. In 20 per cent of the cases, treatment is unsuccessful.

Treatment consists of saline laxatives, mouth rinses with 0.5 per cent Aureomycin solutions and the avoidance of tobacco and hard foods. The individual lesions are touched up with a 10 per cent solution of silver nitrate or alum stick. If a bland diet and a saline laxative are prescribed for a few days the condition usually clears up rapidly. In the recurrent cases, multiple small pox vaccination may be successful although its value is disputed.

ORAL MONILIASIS

(THRUSH)

Thrush consists of a glistening or grayish-white feltlike membrane on the buccal the lingual or the sublingual surfaces. The angles of the mouth, as well as other cutaneous areas also may be affected. Two types are recognized (1) a superficial type which responds to treatment, and (2) a submucous, or deeper variety which is resistant to therapy.

Etiology In infants, thrush may appear in the mouths of bottle fed babies as dry white patches which are easily scraped

off *Candida albicans* which is found in the lesions, may traverse the gastro-intestinal tract and become the source of napkin eruptions. In the aged diabetes, chronic colitis and debilitating diseases may lower the resistance to infection. Prolonged antibiotic therapy by reducing the bacterial flora, may permit an overgrowth of *Monilia* and cause glossitis, stomatitis and seborrhealike lesions of the nasolabial folds.

Diagnosis. The membrane is washed in alcohol and planted on Sabouraud's medium. In all cases, a roentgenogram of the chest should be made to exclude lung involvement. The stools should be examined for *Monilia*.

Differential Diagnosis. Contact stomatitis from dental plates must be considered. Patch tests and a trial period in which the dentures are not used may be helpful. Leukoplakia and lichen planus are ruled out by biopsy studies.

Treatment. When the disease occurs in infants, the nipples of the bottles should be sterilized before each feeding. In most cases a 1 to 5 per cent aqueous solution of gentian violet is effective. The parts should be mopped also with a 10 per cent aqueous solution of sodium caprylate several times a day. If the moniliasis is caused by an antibiotic, it should be discontinued if feasible, and troches of Mycostatin (Squibb) and an aluminum hydroxide preparation prescribed.

Resistant cases should receive a high-caloric diet, liver extract and oral Mycostatin until a satisfactory result is obtained. Locally a 50 per cent solution of trichloroacetic acid may cause desquamation of the lesions.

VINCENT'S INFECTION

(TRENCH MOUTH)

Trench mouth or oropharyngeal fusospirochetosis, is an acute, chronic or latent infection of the mucous membranes especially of the mouth and is characterized by superficial or deep ulceration. The incubation period is unknown.

Clinical Description. In mild cases, a thin membranous film is observed on the gums, the buccogingival sulcus or the buccal mucosa in the vicinity of the last molar teeth. In severe cases, the gums are swollen and tender the sockets of the teeth may be exposed, due to deep ulceration. When the buccal mucosa is affected, the lesions consist of nonindurated superficial ulcerated areas which bleed easily. These are covered with a dirty-grayish

membrane. The edge of the ulcer is ill-defined and is surrounded by a zone of dusky hyperemia.

The severe cases are associated with fever and symptoms of a general toxemia. Enlargement of the draining lymph nodes is the rule. There is often pain with mastication and more or less salivation and halitosis. In extensive cases the palate the tonsils, the larynx and the pharynx may be involved. Cases have been reported also on the mucous membranes of the genitalia.

Etiology The disease is more frequent in young adults and in those with oral sepsis dietary deficiencies oral trauma and debilitating diseases.

The symbiotic organisms, the fusiform bacillus (*Fusiformis dentium*) and the spiral organism (*Borrelia vincentii*) are found in the lesions but definite proof of direct cause is lacking. They are also present in various other conditions in the mouth and the throat, including ulcerative stomatitis, gingivitis, pyorrhea, devitalized areas, caries and tonsillitis. They do not grow on ordinary culture media.

Differential Diagnosis. The condition must be differentiated from mucous patches of syphilis, diphtheria, scurvy, granulocytopenic angina and mercurial stomatitis.

Diagnosis. Smears from the lesion stained with Löffler's alkaline methylene blue reveal the organisms in positive cases. However microscopic diagnosis is of no value unless the characteristic symptoms and signs of the disease are present.

Prophylaxis. This includes separate eating and drinking utensils, proper disinfection of these and a soap dentifrice. The general resistance should be increased with tonics, high-caloric diet and vitamin C.

Treatment. In the mild cases, the daily local application of an aqueous 5 per cent gentian-violet or methylene-blue solution is usually effective. The mouth also should be swabbed or rinsed for five minutes at a time several times a day with equal parts of hydrogen peroxide and water or a sodium-perborate paste applied. Niacin (from 150 to 300 mg daily) is also effective in some cases.

Penicillin is the antibiotic of choice. In mild cases, the lozenge is prescribed every 4 hours, or wet packs of the solution may be applied directly to the area (500 units per cc. of normal saline). In severe cases 300,000 units of procaine penicillin G injected intramuscularly daily for 4 days clears up the average case.

Smoking is contraindicated and dental work in the mouth should not be undertaken until the clinical manifestations have disappeared and until two consecutive smears taken at weekly intervals are negative. A bland diet is important.

DISEASES OF THE TONGUE

VERRUCA

Verruca of the tongue is a rather frequent type of tumor and is acquired sometimes by the habit of biting warts on the hands. After removal it should be examined to rule out carcinoma. These lesions are removed best with the cautery or the high-frequency wire loop.

CHANCRE OF THE TONGUE

This is described under Syphilis (See p 333) They rarely are seen in recent years since the penicillin era has reduced the incidence of early syphilis in this country. Three varieties may occur: the dry hard papule, the superficial erosion and the ulcerated



FIG. 196. Extensive condylooma (verruca) acuminata of the tongue in a man of 75 (From Dr. B. Usher)

papule (hunterian chancre) They are characterized by slow development, painlessness, induration and regional adenopathy

TUBERCULOSIS

Tuberculosis may affect the tongue in patients with far advanced pulmonary or laryngeal cases of the disease. Rarely the infection may be primary (hematogenous) but is usually secondary from local inoculation with infective sputum.

Types, Nodular ulcerative (mouse-eaten or fissured) or verrucous lesions may occur. The lesions are painful and often are covered with a yellowish exudate.

Etiology Trauma from irregular teeth or dentures often favors inoculation.

Diagnosis is made by smears from the lesion, clinical appearance, biopsy, animal inoculation and a study of the lungs or the larynx.

Prognosis is poor since most of the cases occur in patients with far-advanced tuberculosis.

Treatment consists of isonicotinic acid, PAS or streptomycin and general hygienic measures to combat the infection.

BLACK HARRY TONGUE

This rare condition, also called lingua nigra, is characterized by the presence of brownish or black plush in the midline of the dorsum of the tongue, the lesions consisting of piled-up epidermal cells which are unable to desquamate normally. The color is due to sulfides, oxidation or chromogenic bacteria. Three types are recognized, (1) the acquired type from vitamin deficiencies (chronic alcoholics), oral antibiotics, chewing tobacco or sodium-borate toothpastes, (2) the mycotic type from various yeasts and fungi, and (3) the senile type seen in elderly patients with chronic nasopharyngeal infections.

Pathology There is a hypertrophy of the filiform papillae.

Treatment includes improvement of the nutritional status of the patient and polyvitamin therapy. The tongue should be scraped daily to remove food particles and debris. Exfoliation of the filaments with triweekly applications of iodine tincture, 30 per cent trichloroacetic acid or a 10 per cent alcoholic solution of salicylic acid frequently will cure the condition. An antiseptic mouthwash used daily is good prophylactic treatment after ap-

parent cure. In stubborn cases, one x ray treatment (300 r) by an experienced therapist may effect a cure within 10 days.

PURPURA OF THE TONGUE

Purpura of the tongue consists of smooth elevated purplish patches, usually appearing suddenly and failing to disappear on pressure. The condition which is rare, may be caused by drugs (iodides, arsenicals, chloral hydrate and sera) severe anemias, leukemias and hemophilia and toxins (insect bites and fevers).

Treatment is that of the cause. Dental operations should be deferred.

SCROTAL TONGUE

Scrotal (furrowed) tongue is a persistent, familial or congenital condition consisting of superficial and deep grooves on the tongue, together with a slight hypertrophy of the tissue. The surface usually has a glazed appearance. Scrotal tongue predisposes to various types of glossitis.



FIG. 197 Black hairy tongue. Cultures of *Moraxella* and chromogenic bacteria were obtained.

Treatment is ineffective and unnecessary except for ordinary cleanliness to keep the sulci free from food and debris.

PAPILLITIS

("ROUGH TONGUE")

An enlargement of the circumvallate papilla at the base of the tongue may be caused by sharp teeth irritation by cusps of the molars, lower partial dentures or poor hygiene. When the filiform papillae at the tip of the tongue are hypertrophied, there may be more or less pain. The following causes should be eliminated: irritants (alcohol, tobacco, lingual bars, calculus, irritating mouthwashes), psychoneuroses, cancerphobia and habit of biting tip with incisors.

Treatment depends upon the cause. If persistent, light fulguration may be necessary.

BURNING TONGUE (GLOSSOPYROSIS)

This condition is characterized by a burning sensation which may affect the entire tongue or may be limited to the tip. The



FIG. 193. Scrotal tongue.

temporary cases are usually the result of obvious causes the persistent cases often occur in psychoneurotics.

Physical examination is negative except for some hypertrophy of the papillae.

Etiology The following causes should be investigated excessive smoking or drinking vitamin-B deficiencies, anemia, irritating mouthwashes dissimilar metals in the mouth the temporomandibular syndrome, and gastric hypoacidity or hyperacidity. The psychosomatic (cancerphobia and syphilophobia) factor must be investigated in persistent cases. Alvarez* states that in patients over 50 a slight cerebral hemorrhage should be considered.

Differential Diagnosis. Neuritis of the lingual branch of the fifth nerve should be ruled out.

Treatment depends upon the cause. Proper mouth hygiene mild astringent mouthwashes, avoidance of smoking, and vitamin-B₁₂ therapy may be helpful. If the mouth is dry a trial with nicotinamide (50 mg twice daily) or neostigmine bromide (7.5 mg, t.i.d.) is advisable. Psychiatric consultation is necessary in rebellious cases if not benefited by reserpine with its tranquillizing effect, but co-operation is seldom forthcoming.

GEOGRAPHIC TONGUE

Geographic tongue is a disorder of unknown cause characterized by the appearance of smooth, red patches on the tip and the edges, with central clearing and peripheral extension. The border of the patch is raised slightly is lighter in color than the center and has festooned or gyrate edges. The pattern appears to change day by day. Although there is no pain or tenderness, there is a slight sensitivity to acid foods and tobacco. In most cases the lesions occur on the dorsum of the tongue.

Etiology The cause of the peculiar desquamation of the surface epithelium is unknown.

Course. This disease is subject to remissions and exacerbations. Since the condition may last from a few days to a year it is difficult to offer a prognosis.

Differential diagnosis must be made from mucous patches, erythema multiforme and drug eruptions.

Treatment Prolonged Aureomycin therapy or troches of Mycostatin (Squibb) may be successful in some cases. Hot acid

*Alvarez, W. C. *The Neuroses*. Philadelphia, Saunders, 1951.

and coarse foods, as well as tobacco should be prohibited. If anemia is present, liver injections or iron and vitamin-B tonics are useful. No harm can result from prescribing a soothing mouth-wash.

MOELLER'S GLOSSITIS

This uncommon glossitis is characterized by painful circumscribed raw bright red patches. The condition is chronic. It often is cyclic and is more common in women.

The cause of the disease is unknown. There seems to be no relation to pernicious anemia or avitaminoses.

Differential diagnosis is mainly from geographic tongue in which the lesions have a tendency to coalesce pain is absent and duration usually is under 6 months.

Treatment is ineffective although the corticosteroids may be useful.

GLOSSITIS RHOMBICA MEDIANA

This condition consists of a smooth, shiny elevated rhomboidal plaque in the center of the dorsum of the tongue anterior



FIG. 199 Geographic tongue.

to the circumvallate papilla. Since the condition does not disturb the patient, treatment is unnecessary.

SMOOTH ATROPHY OF THE TONGUE

Superficial sclerosing syphilitic glossitis is a sequel to syphilitic infiltration, which leaves an atrophic surface on the tongue. The condition usually involves the entire tongue but occasionally is unilateral. Another type, without sclerosis, is associated with severe anemias and late visceral carcinoma. Differential diagnosis must be made from the bright-red tongue of pernicious anemia. Treatment is that described for late syphilis.



FIG. 200. Granuloma pyogenicum on tongue, following a minor accidental injury. The growth was removed with the cautery.

MISCELLANEOUS CONDITIONS

OCCUPATIONAL CONDITIONS

The mouth and its adnexa may be affected by direct contact or absorption of various fumes, animal, vegetable or mineral dusts, heavy metals, chemicals or radioactive products. The clinical lesions are variable (e.g., gingivitis from bismuth and osteonecrosis from radioactive salts).

ORAL MANIFESTATIONS OF SYSTEMIC DISEASES

Many of the serious metabolic diseases and blood dyscrasias may involve the oral mucous membranes. These include diabetes, pernicious anemia, the leukemias, malignant neutropenia and thrombocytic purpura.

Symptoms. Clinical manifestations include gingivitis ulcerations, bleeding tendency pallor of the mucous membranes and purpuric lesions.

Diagnosis. The presence of any of the above lesions should awaken suspicion. Confirmation is made by the history hematologic studies and the usual tests when indicated.

Treatment should be instituted as soon as the diagnosis is made.

MUCOUS MEMBRANE NEUROSES

Cheek biting glossodynia, cancerphobia, recurrent herpes simplex halitosis, papillitis of the tongue and irritation of the palatine papillae may occur in the nervous, tense type of individual. These neuroses are 10 times more common in women.

Treatment consists of evaluation of the cause, sedation and superficial psychotherapy

PLUMMER VINSON SYNDROME (SIDEROPENIC DYSPHAGIA)

Plummer Vinson syndrome occurs almost exclusively in women from 40 to 50 years of age.

It is listed as a precancerous condition because of the occurrence of malignant degeneration in the atrophic mucosal lesions. The mucous membranes of the tongue the larynx, the pharynx and the upper end of the esophagus are the sites of a chronic inflammation which later is replaced by atrophic changes. Other symptoms are hypochromatic anemia *perleche* *achylia gastrica*, dysphagia, spoon nails (*koilonychia*) and symptoms of a vitamin-B deficiency

Etiology A severe nutritional disturbance or chronic hemorrhages are basic factors.

Differential Diagnosis. Pernicious anemia is ruled out by bone marrow examination, serum iron studies and a vitamin B₁₂ therapeutic test. Anemia of visceral carcinoma may be detected by x-ray studies of the gastro-intestinal tract and finding occult blood in the stools.

Treatment consists of liver vitamin A, ferrous sulfate 15 gr t.i.d., dilute hydrochloric acid and a high-protein diet.

PERIPHYCIOUS

(See p 142)

NURSING ASPECTS

If the mucous membranes of the mouth are involved the physician probably will wish to examine the anogenital mucosae also. The finding of certain lesions may call for an inspection of the entire skin. A good light is important.

The nurse should have the following items available: tongue depressors, clean glass microscopic slides, cotton applicators, culture tubes, pus basins, solutions of 1 per cent gentian violet and methylosanillin, trichloroacetic acid, tincture of iodine and $\frac{1}{4}$ to 1 per cent procaine with epinephrine, a biopsy set and an electrocautery and a fulgurator in good working order. A modified type of dental chair is preferable for examination and treatment of oral conditions.

Special attention should be paid to the presence of foul odor, salivation, local hypersensitiveness, bloody saliva or extreme dryness.

Congenital Disorders

NEVI

VASCULAR NEVI

PIGMENTED AND NON

PIGMENTED MOLES

NEVOID DISORDERS

XERODERMA

ICTHYOSIS SIMPLEX

KERATODERMIA PALMARIS ET

PLANTARIS

NEVOID DISORDERS (*Contd*)

KERATOSIS FOLLICULARIS

KERATOSIS PILARIS

EPIDERMOLYSIS BULLOSA

NEUROFIBROMATOSIS

ADENOMA SEBACEUM

DERMATOSIS PAPULOSA NIGRA

ALBINISM

NURSING ASPECTS

CONGENITAL affections of the skin are fairly common. Practically every individual has one or more nevi. Their importance lies in the fact that they are cosmetically objectionable occasionally they undergo malignant degeneration. The extensive types may be associated with other developmental ectodermal defects, including anomalies of teeth, tongue hair and nails cho-roid changes, harelip cleft palate, clubfoot and webbed fingers. A nevus is a localized anomaly while generalized conditions are described as nevoid.

The skin and the nervous system are derived from a common embryonal structure. Therefore, it is not unusual for nerve and cutaneous anomalies to coexist (neurofibromatosis, adenoma sebaceum, angiomas of the brain, unilateral hypertrophy etc.) Mental deficiency is often present in extensive nevic conditions.

These disorders are congenital or hereditary. If they are not clinically manifest at birth the fundamental defect exists and may become activated at puberty or later in life (nevus tardus).

Some congenital conditions pathologically are hyperplasias, others are hypoplasias. Epidermis, blood vessels, lymph vessels, sweat and sebaceous glands, hair follicles, cutaneous nerves, fibrous and elastic tissues may share in the process. Generally speaking, they are all hereditary defects of the germ plasm and

are dominant, recessive or sex linked characteristics.* Whether they result from constitutional or environmental factors, physiochemical changes or imperfect implantation of the fertilized ovum is unknown.

NEVI

A nevus is a skin lesion resulting from an embryonal tissue disturbance, the objective manifestations of which may be present at birth or may appear at any time thereafter. Moles are examples of these benign neoplasias and represent the most common form of nevus growth †

Classification

I. VASCULAR NEVI

- 1 *Blood vessel origin* (1) flat port wine mark (2) raised, or "strawberry" hemangioma (3) cavernous hemangioma (4) sessile, or spider nevus.
- 2 *Lymphatic origin* (1) lymphangiectasis (2) lymphangioma.

II. NONVASCULAR NEVI

- 1 *Soft nevi* (moles) (1) nonpigmented (2) pigmented (3) hairy (4) giant nevus.
- 2 *Hard nevi* (1) verrucous (2) linear (3) ichthyosis hystrix.
- 3 *Fibromatous nevi* (1) fibroma molluscum (Recklinghausen's disease) (2) nevus lipomatodes.
- 4 *Glandular nevi* (1) adenoma sebaceum (2) syringocystadenoma.

VASCULAR NEVI

An angioma is a congenital localized hyperplasia of pre-existing vessels in the skin. The majority disappear at adolescence unless they are of the deep variety or are mixed with lipomatous elements.

The theory common among the laity that these are caused by prenatal maternal impressions is false, of course.

Therapy depends upon location, size, growth characteristics, and histologic structure.

*The inheritance of the congenital dermatoses is discussed in Cockayne E. A. *Inherited Abnormalities of the Skin and Its Appendages*, New York, Oxford, 1933.

†Webster J. R. The identification and management of the more common nevi, *M. Clin. North America* 33 219-234, 1949.

Strawberry Nevus

Strawberry nevi are slow-growing raised bright-red or purple soft tumors. Usually noticed within a few days after birth, they may be mistaken for a bruise by the mother. They are partly cutaneous (red) and partly subcutaneous (purple). They are usually the size of a quarter but may be larger. Most of the cases



FIG. 201 Strawberry nevus (on buttock) undergoing spontaneous involution.

occur on the head or the neck, occasionally they appear on the mucocutaneous surfaces of the nose, the lip, the vulva or the anus, with resulting ulceration as a rule. In some cases the nevus may disappear spontaneously or as a result of trauma. Hemorrhage rarely occurs, even if the lesions are traumatized. They consist of immature blood vessels, which fortunately are sensitive to radium and sclerosing solutions. The younger the patient, the better the therapeutic result.

Pathology. The normal capillaries in the upper dermis are dilated. Newly formed blood vessels are also present. Proliferation of the connective tissue is likewise a feature. The epidermis is thinned out by the upward pressure of the vascular hyperplasia.

Course. Most cases will disappear spontaneously by the first year, leaving slight atrophy or a patch of dilated tiny capillaries as a faint red reminder of the angioma. In some lesions, central involution appears early, leaving a peripheral ring of angiomatous tissue.

Treatment. If untreated, the lesion becomes more radio-resistant with the passage of time, fibrous changes may occur

or ulceration with deep scarring may result. In any case, the parents should be told that spontaneous healing and all therapeutic measures may result eventually in a certain degree of atrophy.

If skilled consultation is available it is best to treat the lesion since most parents regard these lesions as stigmata.

BETA RADIUM is the preferred method of treatment because of the ease of application and the excellence of results. Since these lesions are more sensitive to radiation during early infancy treatment should be instituted before the age of 6 months if possible. A half-strength radium-element plaque screened with 0.1 mm. aluminum, is applied to the lesion for from 10 to 20 minutes. Treatment is repeated in 6 weeks if the reaction from the previous application has disappeared. Usually from 2 to 4 applications suffice for the average case. Overtreatment must be avoided.

CONTACT X RAY THERAPY is also effective in skilled hands.

SCLEROSING SOLUTIONS OR SOLID CARBON DIOXIDE are indicated in the treatment of lesions on the scalp, the genitalia, the breasts or over the epiphyses, where radium therapy might cause permanent damage to the underlying structures.

EXCISION is indicated in cases complicated by radiodermatitis or fibrous tissue.

Ulcerated lesions require no treatment except antibiotic ointments to reduce the chance of infection.

Heimangioma Simplex

This type of angioma is often seen in newborn infants as a dull, red flat blotch over the glabella, the occiput and the eyebrow regions. Crying intensifies the color. No therapy is indicated since the lesions usually disappear by the end of the first year.

A nuchal type which persists into adult life (*Unna's nevus*) is observed in about 5 per cent of the population.

Cavernous Angioma

These are usually large, round or flat, lobulated or nodular deep lesions. They often are found on the head and the neck. In most of the cases they are more subcutaneous than cutaneous. If untreated they often become fibrous. These lesions can be very disfiguring.

Differential diagnosis is rarely necessary but an arteriovenous aneurysm (traumatic) which pulsates under the examining finger may cause confusion.

Treatment. Small lesions should be injected with an amount of sclerosing solution equal to one half the volume of the lesion every 2 to 4 weeks. Isolated fibrous lesions should be removed by surgical methods (resection or enucleation). Deep lesions which appear to be surgical risks are treated best with interstitial gamma radium applications.

Port-wine Angiomas

Port-wine angiomas are large, unilateral or segmental, flat uneven, dull-red or violaceous lesions. The surface may be smooth or verrucous. They usually occur on the side of the face or the neck, occasionally on the back of the hands. The superficial types blanch on diascopic pressure the deep type does not.

Pathology. These hypertrophic growths consist of dilated capillaries, endothelial spaces and loose connective tissue. They may be associated with other nevoid conditions angiomatosis of the meninges and the brain sometimes is present in the extensive cases involving the trigeminal area.

Differential diagnosis is made from arteriovenous aneurysm when on an extremity by the increased warmth and the perspiration when compared with the normal one.

Prognosis. Port wine marks rarely disappear spontaneously but, in later life, many become fainter and only a telangiectatic network remains. They are usually permanent because of their deep-seated nature. Ulceration and hemorrhage may result from trauma or unskillful treatment. They are notoriously difficult to eradicate.

Treatment. Since the usual and the unusual methods of destruction only add to the disfigurement, the best treatment is to leave them alone. Thorium X may be useful in selected cases and may be obtained in alcohol lacquer or ointment form. The average dose is 150-300 microcuries (equal to 1000-2000 electrostatic units). Applications are made at 4-week intervals. Cover mark, a proprietary enamel, or skilled tattooing with special pigments may partially conceal the condition.

Hemangioendothelioma

This is a rare nodular angiomatous tumor usually occurring in adults. It bleeds easily and often follows trauma.

Treatment involves excision of the growth.

Telangiectasia

These are dilated pre-existing capillaries or new formations. The congenital varieties include Rendu-Osler disease, spider nevi, senile angiomas and telangiectasis associated with adenoma sebaceum.

Spider or stellate nevi (nevus araneus) are dilated capillaries resembling tiny red spiders that radiate from a central arteriole. They are common on the upper half of the face. They usually appear spontaneously but may follow slight trauma. They also may occur during pregnancy and sometimes after prolonged corticotropin therapy and a pulsating type is found in some cases of cirrhosis of the liver. These lesions are destroyed easily by electrolysis. The platinum needle is inserted into the central vessel which when destroyed causes the feeding vessels to atrophy.

Multiple hemorrhagic familial telangiectasia (Rendu-Osler disease) is a rare familial and hereditary disorder characterized by recurrent nosebleeds and telangiectasis of the skin and the mucous membranes. The disease usually begins in infancy when punctate and stellate, dilated capillaries appear on the face, the trunk, or the mucous membranes of the mouth. Bleeding from the nose is the most common type of hemorrhage although hematemesis, rectal bleeding and metrorrhagia have been noted also. Bleeding and clotting time, fragility tests, blood-calcium and blood-platelet count are normal. A secondary anemia is usually present.

Etiology The condition is a dominant familial and hereditary disorder. It may be transmitted by both sexes. An absence of elastic tissue or decreased elastic tissue in the vessel walls and the surrounding tissues is the probable cause of the disturbance.

Differential diagnosis is made from purpura hemorrhagica and hemophilia by the history and blood testing, also from pulsating telangiectasia associated with hepatic disease.

Course. If not controlled, a severe secondary anemia results. Death has occurred from fatal epistaxis.

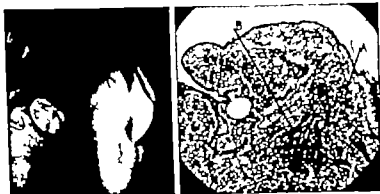


FIG. 202 (Left) Pedunculated nonpigmented mole. FIG. 203 (Right) Pigmented nevus with (A) deposit of melanin and (B) cords of nevus cells.

Treatment. Internal bleeding can be controlled by injections of Thromboplastin, oral Fibrinogen or Ceanothyn. Bleeding from the nose can be controlled by packing styptics, thrombin U.S.P., Stypven (Burroughs-Wellcome) Thrombol (Sharpe & Dohme) or absorbable gelatin sponge U.S.P. Oral telangiectasia should be destroyed by electrodesiccation or the electro-cautery. Blood transfusions are necessary in those cases with prolonged blood loss and secondary anemia.

Senile Angiomas (Ruby Spots)

These are pinhead-sized, soft red lesions which often are observed on the trunk in middle-aged and elderly individuals.

Differential diagnosis. Pulsating angiomas associated with cirrhosis of the liver and spider nevi with radiating tiny capillaries must be considered. No treatment is indicated, although a light touch with the electro-cautery is all that is necessary to destroy them.

Lymphangiomas

Lymphangiomas are divided into three types (1) simplex, (2) hypertrophic and (3) cavernous. The latter two often are mixed with angiomatous, fibrous or lipomatous elements.

Lymphangioma simplex is a diffuse, painless swelling of the lip, the tongue or the genitalia. A few straw-colored vesicles may be present. Treatment is unsatisfactory.

Hemangioendothelioma

This is a rare nodular angiomatous tumor usually occurring in adults. It bleeds easily and often follows trauma.

Treatment involves excision of the growth.

Telangiectasia

These are dilated pre-existing capillaries or new formations. The congenital varieties include Rendu-Osler disease spider nevi, senile angiomas and telangiectasis associated with adenoma sebaceum.

Spider or stellate nevi (nevus araneus) are dilated capillaries resembling tiny red spiders that radiate from a central arteriole. They are common on the upper half of the face. They usually appear spontaneously but may follow slight trauma. They also may occur during pregnancy and sometimes after prolonged corticotropin therapy and a pulsating type is found in some cases of cirrhosis of the liver. These lesions are destroyed easily by electrolysis. The platinum needle is inserted into the central vessel which, when destroyed causes the feeding vessels to atrophy.

Multiple hemorrhagic familial telangiectasia (Rendu-Osler disease) is a rare familial and hereditary disorder characterized by recurrent nosebleeds and telangiectasia of the skin and the mucous membranes. The disease usually begins in infancy when punctate and stellate, dilated capillaries appear on the face the trunk, or the mucous membranes of the mouth. Bleeding from the nose is the most common type of hemorrhage although hematemesis, rectal bleeding and metrorrhagia have been noted also. Bleeding and clotting time fragility tests, blood-calcium and blood-platelet count are normal. A secondary anemia is usually present.

Etiology The condition is a dominant familial and hereditary disorder. It may be transmitted by both sexes. An absence of elastic tissue or decreased elastic tissue in the vessel walls and the surrounding tissues is the probable cause of the disturbance.

Differential diagnosis is made from purpura hemorrhagica and hemophilia by the history and blood testing also from pulsating telangiectasia associated with hepatic disease.

Course. If not controlled a severe secondary anemia results. Death has occurred from fatal epistaxis.

nevus then can be removed by excision or destroyed with light fulguration or superficial applications of the cautery. Hypertrophic scars, or even keloids, may follow any electrosurgical method of removal.

Raised Pigmented Nevus

Raised pigmented nevus which contain nevus cells produced by benign proliferation of melanoblasts, include circumscribed black, bluish or brown moles. They are important lesions and are potentially dangerous because of the possibility of degeneration into malignant melanoma. They are usually smooth and hairless and are found anywhere on the body. For signs indicative of malignant changes, see page 397.



FIG. 204. Raised pigmented nevus.

Pathology. Pigmented hairy and warty nevi, depending on their position in the epidermis, are classified as (1) intra-epithelial (2) junctional (dermo-epithelial) type, (3) intradermal and (4) mixed. The junction type (epidermodermal) may be the forerunner of melanoma or nevocarcinoma if irritated.

Diagnosis. The history (duration as long as the patient can remember) clinical appearance (no increase in size or color) and biopsy in all cases establish the diagnosis.

Differential diagnosis. Pigmented keratoses and pigmented basal-cell cancer may resemble the pigmented mole occasionally.

Treatment. These nevi may be removed if located on pressure areas, or if they are cosmetically or psychologically objectionable to the patient. Surgical excision with the knife is the method of

choice. X ray radiation solid carbon dioxide electrolysis and caustics are dangerous. It is important to make routine sections of all moles removed by excision. When bleeding crusting or rapid growth occurs, immediate radical excision is indicated.

Multiple small pigmented nevi should be left alone. Single large pigmented nevi in infants, if cosmetically undesirable should receive the attention of the plastic surgeon.*

Linear Nevi

Linear (systematized) nevi are rare congenital lesions which vary widely in their structure and appearance. They are unilateral growths which often appear along lines of cleavage or along the course of a nerve trunk, or a blood vessel. In some cases they are more widely distributed, with scattered lesions involving one half of the trunk and the face. The mucous membranes of the mouth the tongue or the genitalia also may be involved. Clinically the lesions may be ichthyotic pigmented psoriasiform lichenoid verrucous, keratotic, comedoniform or papillomatous.

Etiology Remnants of dermal tissue in embryonic fissures or in fragile zones (Caro and Senear) appear to be a plausible explanation.

Differential Diagnosis. Linear psoriasis, linear lichen planus, lichen striatus and ichthyosis hystrix have a characteristic histologic picture history and clinical features.

Treatment. Surgical treatment is indicated except in the extensive cases which are better left alone. In the superficial type surgical planing may result in a satisfactory cosmetic appearance.

Mongolian Spots

Mongolian spots are congenital bluish macules, single or multiple usually found in the dark races and often found at birth over the sacral region. They are of mesodermal origin with melanoblasts in the corium but do not degenerate into melanoma. The "mongolian cell" is a long fusiform melanoblast having several dendritic processes scattered without clumping

For further discussion of the diagnostic and therapeutic problems of the pigmented nevi, see Becker, S. W. Diagnosis and treatment of pigmented nevi, *Arch. Dermat. & Syph.* 60:44 1949. Swerdlow, M. Nevi: Problems of misdiagnosis, *Am. J. Clin. Path.* 22:1054 1952 and Frank, S. B. Management of pigmented nevi, *A.M.A. Arch. Dermat. & Syph.* 69 172 187 1954.

between the elastic and the fibrous tissue fibers.* These lesions disappear in from 2 months to 8 years.

Blue Nevus

Blue nevi are rare oval, light blue macules of mesodermal origin. The dark bluish nevus cells are found deep in the cutis. They sometimes occur on the face or the scalp. They are considered to be persistent Mongolian spots. Clinically they are difficult to distinguish from the blue-black moles. Treatment consists of wide excision if the lesion is subject to irritation and if the diagnosis is confirmed by microscopic study



FIG. 204 A. Congenital xeroderma with definite scaling always associated with pruritus in winter

NEVOID DISORDERS

XERODERMA

Xeroderma is the mildest form of ichthyosis and consists of a dryness and a mild scaliness of the entire cutaneous surface. The condition is more prominent on the trunk and the extensor surfaces and the palms and the soles. A moderate degree of ker-

* For a discussion of the subject, see Cole, H. V. J. et al. Persistent aberrant mongolian spots, Arch. Dermat. & Syph. 61:244 1950.

tosis pilaris may be present. The condition exists from birth or appears later in life and may be familial. Superfatted soaps, small doses of thyroid, vitamin A and oily lotions after the bath are indicated.

ICHTHYOSIS SIMPLEX

Ichthyosis simplex ("fish-skin disease") is a common congenital or inherited usually persistent, scaly condition of the skin. The skin is dry, harsh rough and covered with thin dirty-gray or brownish branny scales. There is a lack of flexibility and elasticity, with a tendency to fissure formation over the joints. Follicular hyperkeratoses are often present on the extensor surfaces of the extremities and the buttocks.

Ichthyosis is always universal in its distribution. There is little or no itching except in the wintertime when the condition is aggravated by cold weather and decreased secretions.

An ichthyotic skin is vulnerable to irritation from various external agents. Eczematization is apt to occur from mild irritation. Patients with ichthyosis should be warned of the danger of coming in contact with irritants or of assuming occupational hazards.

Pathology Hyperkeratosis is present without parakeratosis. Strangely the granular layer is thinned or absent. The prickle-cell layer is flattened, while the pressure of the hyperkeratosis in the follicular openings causes some atrophy of the sweat glands and the oil glands.

Course. Mild cases may clear up spontaneously at puberty. The more extensive cases become stationary at adolescence. Cure is impossible, but the disorder can be controlled.

Treatment. Internal treatment is of little or no value although large doses of vitamin A are apparently effective in some cases, even if the serum vitamin A levels are within normal limits. Thyroid medication is only of temporary benefit but should not be prescribed without a basal metabolism test and a chest examination for cardiac and pulmonary disease.

LOCAL TREATMENT If persisted in, will ameliorate the condition. A daily hot bath using tincture of green soap and moderate friction is necessary to reduce the scaling. This should be immediately followed by inunction with vitamin A ointments, toilet lanolin or 2 per cent salicylic acid in olive or cotton seed



FIG. 205 Hereditary keratoderma palmaris et plantaris
in mother son and daughter

oil. If eczematization occurs, the application of calamine liniment for several days followed by an emollient ointment is indicated. Warm clothing is essential during the winter months.

ERYTHRODERMIA ICTHYOSIFORME CONGENITALE

(See p. 475)

KERATODERMIA PALMARIS ET PLANTARIS

This condition is a congenital or an hereditary diffuse or punctate hyperkeratosis of the palms or the soles. The disorder is usually noted first at birth as a persistent hyperemia. The hyperkeratotic condition becomes noticeable at puberty or soon thereafter. The palmar and the plantar surfaces are dry, rough, thickened, fissured and definitely hyperkeratotic. There is a lack of pliability and elasticity. The borders of the hyperkeratoses, which exhibit a pinkish halo end abruptly at the wrists.

Etiology There is a definite hereditary history in the majority of the cases.

Pathology All the layers of the skin share in the hyperkeratotic process which is usually more pronounced in the vicinity of the glandular orifices. There is moderate dilatation of the blood vessels in the corium.

Course. The disorder is persistent, but, with emollient treatment, the skin usually can be kept in fair condition.

Differential Diagnosis. Occupational callus, arsenical keratoses, hyperkeratotic type of epidermophytosis and psoriasis, must be considered.

Treatment. The use of 40 per cent salicylic-acid plasters and Whitfield's ointment offers temporary relief. When pain and interference with function is present, plastic surgery (pedicle flaps from the thighs to replace the involved palms or soles) may be considered.

KERATOSIS FOLLICULARIS

Keratosis follicularis (Darier's disease) is a rare generalized nevus disorder which develops before puberty and consists of a symmetrical eruption of follicular hyperkeratoses. The primary lesion is a small red or brown papule, with a surrounding halo of faint erythema around the pilosebaceous opening not unlike a flat juvenile wart. These later become discrete and confluent papillomatous papules. As the lesions coalesce to form patches of various sizes, they become brown, greasy and crusted. The crusts are hard and dry and have elongations on their undersurface which dip down into the papules. Vegetating masses develop in the moist regions, including the genitocrural the postaural the nasolabial and the axillary regions. The scalp often is covered with greasy scales or crusts. Favorite sites are the seborrheic regions. The palms the soles and the dorsal surfaces of the fingers may be involved also. Mild to extreme itching may be present. The general health of the patient is not affected. The condition is usually worse during the summer months.

Etiology. The cause of the disorder is unknown, but the following theories have been advanced (1) light sensitivity since sunlight aggravates the condition and (2) a possible form of congenital hypovitaminosis A although few cases respond to vitamin A therapy.

Diagnosis. The clinical character of the lesions, localization, chronicity and biopsy studies make the diagnosis fairly simple. Serum-carotene vitamin A determinations and the dark adaptation tests may be normal.

Pathology. This disease belongs to the dyskeratoses. The presence of the "bird's-eye" corps ronds or degenerated, isolated prickly cells which become separated from one another and intra-

dermal clefts (lacunae) resulting from acantholysis, are characteristic. The pilosebaceous funnels may be dilated and contain horny plugs and degenerated cells.

Course. The disease is progressive and chronic.

Differential diagnosis is mainly from pityriasis rubra pilaris and familial benign pemphigus.

Treatment. There is no known cure for the condition. The crusting can be controlled by the application of oily lotions (e.g. Nivea Oil or Lotocreme). Vitamin A in large doses (150,000 units daily) preferably as the water soluble Aquasol A, combined with hydrocortisone for several months is worth a trial but only a small percentage of the cases show a favorable response. Light therapy including use of x-rays, is contraindicated.

KERATOSIS PILARIS

(FOLLICULAR ICHTHYOSIS)

Follicular ichthyosis is a common congenital hypertrophic disorder of the epidermis, consisting of groups of acuminate, rough, hard, horny papules situated at the mouths of the hair follicles. They are usually the color of the skin but may be gray or light brown. They have a "nutmeg-grater" feel when the finger is rubbed over the lesions. The usual sites are the extensor surfaces of the thighs, the arms, the forearms, the buttocks, and the backs of the legs, practically never on the trunk. The lesions are profuse but discrete and never form patches. Each papule is pierced by a lanugo hair or a broken-off hair. Cold weather aggravates the condition.

Etiology. The disease is congenital, sometimes hereditary or familial. The rate of keratin production in predisposed persons is affected by vitamin and endocrine deficiencies, cold weather and lowered metabolism resulting in the formation of the follicular plugs.*

Pathology. A localized hyperkeratosis is present about the mouths of the pilosebaceous orifices. The lower part of the follicle and its sebaceous gland are atrophic.

Course. The disease is persistent but is hardly noticeable in warm weather.

Differential Diagnosis. CURTIS ANSERINA (goose skin) is a normal physiologic response to exposure to cold. It is nonacutely and evanescent.

*Forman, L. Keratosis pilaris, Brit J Dermat 66:1279 1954

LICHEN SCROFULOSORUM consists of coin-sized, round or oval patches of fine scaly papules in children or young adults. The eruption is characterized by slow evolution. There is often evidence of visceral tuberculosis.

THE FOLLICULAR SYPHILID usually occurs on the trunk, the arms and the thighs and is often associated with syphilitic

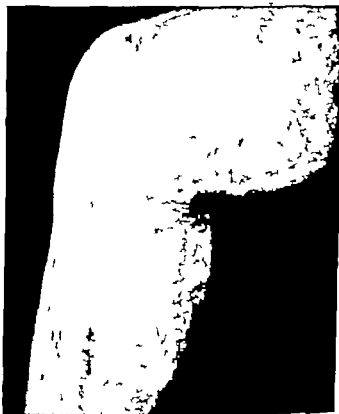


FIG. 206. Keratoma pilaris.

alopecia and a positive serology. The lesions are crowded or grouped dull-red or light brown follicular papules.

Treatment. Infrequent baths are advisable. Daily applications of cocoa butter or an ointment containing 4 per cent precipitated sulfur and 2 per cent salicylic acid are usually effective.

Vitamin A is prescribed as a routine measure. Thyroid extract is indicated in those with low metabolic rates.

EPIDERMOLYSIS BULLOSA

This is a rare nevoid disturbance of the skin and the mucous membranes characterized by a vulnerability to trauma and the presence of bullae over irritated areas.

Types. THE SIMPLE TYPE usually appears on the palms and the soles as flaccid bullae containing clear serum. Some of the lesions are hemorrhagic. The eruption becomes more generalized as the patient grows older. As the old bullae fade they are replaced with thin atrophic scars or pigmented areas. The lesions on the hands are apt to be secondarily infected.

THE DYSTROPHIC TYPE consists of flaccid bullae and dystrophic changes. The nails may be shed and replaced with scar tissue. Dental dystrophies, contractures, scarring and keloidal miliumlike epidermal cysts on the fingers, the elbows and the knees are often present.

Etiology The disorder is often hereditary and familial. Various theories have been advanced, none satisfactory. The hyaluronidase enzyme system may be adversely affected in these cases. Uroporphyrin has been found in some of the congenital cases, but its significance is not thoroughly understood.

Pathology In a small proportion of the cases there is fragmentation or disappearance of the elastic tissue of the corium which may be the result, and not the cause of the disorder. A lack of adhesion between the epidermis and the corium with serous exudation is present in all the cases.

Course. Although the disease is persistent, the vulnerability of the skin tends to decrease after the third decade.

Differential Diagnosis. Hydrom aestivale should be eliminated by direct-light and ultraviolet light studies. bullous porphyria, by urine studies. In infants, bullous syphilis, impetigo and congenital defects of the skin must be considered.

Treatment. Large doses of vitamin C have been reported to be useful but these reports are unconfirmed. Hydrocortisone may control the development of new bullae temporarily. As in ichthyosis, the patient should be taught to live with his skin and to avoid trauma as much as possible. The dystrophic type may present a difficult problem to the plastic surgeon because of the fragility of the tissues.

NEUROFIBROMATOSIS

Multiple neurofibromata or Recklinghausen's disease is an uncommon generalized nevroid disorder which consists of numerous fibromas, pigmented macules, nerve tumors and sometimes visceral anomalies and bone changes. Neuromata may involve the ciliary and the optic nerves.

The disorder begins in childhood or at puberty. The skin gradually becomes studded with numerous growths from the size



FIG. 707 Neurofibromatosis.

of a millet seed to that of a grapefruit. The growths are soft or hard sessile or pedunculated round or oval. They are flesh-colored brown or red in color painful or painless. Some of the nodules are situated along the nerve trunks. The mucous membranes, including the tongue, may be involved also. Included in the cutaneous picture, are one or more pigmentary macules of various sizes and shapes, some, coffee-colored and others, bluish, dark brown or black. Those rare cases consisting of macules only are designated as abortive or *forme fruste* types. Many cases are associated with bone lesions which may consist of (1) erosive defects, (2) scoliosis, (3) disorders of growth (4) bowing and

pseudo-arthritis, (5) intra-osseous cystic lesions and (6) various congenital anomalies. A low physical and mental habitus has been noted but this is neither constant nor diagnostic.

Course. The condition is persistent throughout life. There is always danger of sarcomatous degeneration (13 per cent) in the deeper lesions but rarely in the cutaneous fibromas.

Etiology Very few of the cases are hereditary or familial. The disorder is probably due to intra-uterine changes in the germ plasma.

Pathology The disorder is the result of a widespread nevroid condition involving the nervous, the connective-tissue and the osseous systems. The fibrous tumors are probably the result of a hypertrophy of misplaced embryonal nerve cells. Some of the growths contain terminal nerve fibrils.

Differential diagnosis is from leprosy multiple lipomas and sarcoma. Albright's syndrome consists of one or more ipsilateral pigmented patches, disseminated osteitis fibrosa and sexual precocity in the female.

Treatment. The large pendulous tumors, or myxofibrolipomatous growths, should be surgically removed for their cosmetic effect and to prevent degenerative changes. A physical examination, including roentgenograms of the chest and the bones, should be made annually to detect malignant changes in the lesions.

ADENOMA SEBACEUM

Adenoma sebaceum is a rare nevroid syndrome affecting the central portion of the face involving the sebaceous glands. There are three clinical varieties, (1) the red, or Pringle type, (2) the yellow or Balzer variety and (3) the warty type (Hallopeau and Leredde)

The condition begins in early childhood as a symmetrical eruption of pinhead-sized to pea-sized, pink, red or yellow discrete papules on the flush areas of the face and the nasolabial folds. The surrounding uninvolved skin, as well as the surface of the lesions, are traversed by numerous fine dilated capillaries. In some cases, neurofibromatosis and other nevroid conditions are present. Rarely grayish-white raspberry retinal tumors may be present.

THE PRINGLE SYNDROME consists of three disorders: adenoma sebaceum, (2) tuberose sclerosis, with or without



FIG. 208. (*Left*) Periungual and subungual fibromas of the nails in the adenoma sebaceum syndrome. (*Right*) Adenoma sebaceum with involvement of the flesh areas of the face and the nasolabial folds. (Both from Dr. C. C. Barrett)

discs, and (3) multiple subungual and periungual fibromas of the fingers and the toes.

Etiology The syndrome usually is found in feeble-minded and idiotic children but the skin lesions may be present in normal individuals. There is a familial or inherited tendency in some cases.

Pathology Congenital visceral anomalies (teratomas, etc.) and brain tumors (tuberose sclerosis, which is characterized by potato-like hardenings) are not uncommon findings. Sections of the nodules show a hypertrophy of the sebaceous glands and the surrounding blood vessels, with or without the presence of nevus cells.

Diagnosis. The triad of symmetrical, reddish nevoid nodules, epileptic attacks and nevoid plaques is confirmatory. Encephalograms and x ray pictures of the skull (parietal areas above the level of the lateral ventricles) may be used to determine cerebral pathology.

Differential diagnosis is from cystic acne, colloid milium and multiple benign cystic epithelioma.

Course is slow and persistent.

Treatment. The adenomas can be destroyed by carbon-dioxide snow fulguration or electrolysis followed by surgical planing for the residual scarring.

MULTIPLE BENIGN CYSTIC EPITHELIOMA

(See p 363)

DERMATOSIS PAPULOSA NIGRA

This is a common nevus disorder in Negroes. The eruption consists of small hypertrophic black rounded papules which are grouped on the cheeks, the forehead and about the eyes. The condition is probably a development defect of the pilosebaceous apparatus. Treatment is seldom desired.

SYRINGOCYSTOMA

(See p. 535)



FIG 209 Albinism.

URTICARIA PROMENTORA

(See p. 461)

JUVENILE ACANTHOSIS NIGRICANS

(See p. 459)

ALBINISM

Albinism is a congenital absence of pigment which may be partial or universal. The universal types are usually familial and consist of a total absence of pigment in the skin and its appendages. Albinos have pink irides and red pupils and suffer from vasomotor disturbances, photophobia and nystagmus. The hair is white and silky and the skin of the face has a pinkish hue. Prolonged exposure to the sun or to ultraviolet light results in serious burns, due to the absence of the protective pigment granules.

Treatment is unavailing. Light screening ointments containing 15 per cent *p*-aminobenzoic acid may reduce the danger of sunburn. Tinted glasses may control the photophobia.

NURSING ASPECTS

Congenital defects may be localized or extensive so that the entire cutaneous surface must be examined. Some cases require complete blood counts, basal-metabolism tests, porphyrin and vitamin-A determinations and biopsies.

Infants with ulcerative or bullous conditions should be placed in padded cribs to avoid trauma. Nutritional requirements must be observed if large areas of the skin are involved in the pathologic process.

Dermatologic Therapeutics

TYPES OF LOCAL MEDICATION

PHARMACEUTIC

PREPARATIONS

THE SPECIFIC ACTION OF

DRUGS

SYSTEMIC THERAPY

PHYSICAL METHODS

CRYOTHERAPY (COLD)

PHYSICAL METHODS (*Contd*)

ABRASIVE THERAPY

ELECTROTHERAPY

THERMOTHERAPY

IONTOPHORESIS

PHOTOTHERAPY (LIGHT)

GENERAL THERAPEUTIC

SUGGESTIONS

SUCCESS In the treatment of skin diseases, the correct diagnosis having been made, depends upon the following (1) the co-operation of the patient, (2) the proper choice of medication, (3) the adjustment of the therapy to fit the particular patient, (4) continued observation to detect intolerance, sensitization to medication side reactions or change in the course of the disease and (5) the personality of the physician his interest and his enthusiasm.

Principle of Prescribing Acute subacute and chronic stages of an eruption each require a separate approach so that clinical examination under ideal conditions is very important. When prescribing specific measures, the physician should be guided by two principles. (1) selection of a method or a drug of first choice and (2) the consideration of the site of the lesion or the eruption as influencing that choice. A method or a drug of first choice is the simplest and the most effective one available. Consideration of the site will influence the physician to use judgment in employing irritating drugs on areas where the skin is thin or sensitive.

Untoward results of medication include (1) irritation (2) scarring (3) discoloration from tar salubrins etc. (4) freckling or tanning from x ray or ultraviolet radiation (5) toxic absorption of drugs used locally (6) exfoliative dermatitis in susceptible

Individuals (7) autosensitization (8) drug eruptions (9) drug-fastness (10) grease reactions (flare-ups from an increase in local surface temperature) (11) cross-sensitizations and (12) malign-nant stimulation.

The following rules are useful in prescribing

1 The patient must accept the diagnosis and his co-operation must be obtained.

2 The therapy and the type of dressings must be suitable for ambulatory use.

3 If the method of application the use of more than one preparation or type of bath seems to confuse the patient, detailed instructions should be typewritten.

4 The preparation should be available in one of the local pharmacies not too expensive for the patient's purse stable and cosmetically acceptable.

5 If the patient gives a history of having a sensitive skin only a small amount of the preparation should be prescribed and used on a small area to determine sensitivity Know the state of the patient's skin.

6 When prescribing for patients with infantile eczema atopic eczema and rapidly spreading eruptions, the patient should be requested to notify the doctor within 24 hours if any irritation develops.

7 When prescribing oral antihistaminics, antibiotics or corticosteroids request the patient to notify you at once if side reactions occur

8 The patient should be told how long to use the preparation how to remove it and when to return for the next consultation.

9 Mix your prescription with thought. Do not depend on the alluring ads or the enthusiastic appeals of the detail man.

10 If you can not recognize the disease or make a diagnosis, always treat using physiologic principles that have stood the test of time Treat the stage of the disease not the name of the disease.

11 Do not change medication every 2 or 3 days if it seems to appear beneficial. Give it a chance.

12 Inform the patient at the time of the first visit the cause of the disease (simple terms) the approximate length of time to cure it whether the condition is contagious or not and whether he may continue with his occupation

13 Evaluate all the factors indirectly or directly responsible for the eruption and correct, treat or modify each of them

Hospitalization. Patients with extensive, refractory, spreading or extremely pruritic dermatoses should be hospitalized in order that rest, consultations, surgical procedures, proper medication and attention can be obtained. Hospitalization is advised also for cases requiring intensive investigation, ACTH therapy laboratory studies and for patients unable to comprehend or follow instructions. Dermatologic therapeutic procedures can be managed properly only by nurses trained by dermatologists.

Masterly Inactivity consists of doing nothing except to supply the patient with encouragement and to overcome his anxiety. Treatment should be withheld in patients who are unduly sensitive to all medication in cases under observation in cases of simple drug eruptions and in patients of the faultfinding type.

Nervous tension, financial and domestic problems, chronic fatigue and worry are important predisposing factors in certain types of urticaria, dyshidrosis, rosacea, pruritus, vitiligo alopecia areata, localized neurodermatitis and atopic eczemas. These factors should be handled with tact understanding and charity.

New Drugs. In general, it is unwise for the general practitioner to use the newer drugs in dermatologic conditions until at least 1 year after they have been on the market. By that time their therapeutic value side reactions and sensitizing properties have been studied thoroughly and their indications evaluated.

TYPES OF LOCAL MEDICATION

Success in local treatment depends upon (1) a selection of the proper drug, (2) proper strength of the medication adjusted to the patient's tolerance, (3) proper vehicle and (4) proper method of application. The knowledge of the use of the proper preparation can be gained only by experience.

Indications for Topical Therapy Based on Type of Eruption

Pruritus without eruption	powders
Macular or erythematous	drying lotions
Erythema plus noninflammatory edema	cold aluminum acetate packs
Erythema plus inflammatory edema	hot boric acid packs
Superficial scaly eruptions	mild ointments
Thick scaly eruptions	tar ointments
Excoriated dry eruptions	hydrocortisone ointments
Noninflammatory scaling	oily lotions
Localized vesicular eruptions	pastes
Generalized vesicular eruptions	drying lotions

Bullous eruptions (unruptured)	powders
Bullous eruptions (ruptured)	petroleum gauze dressings
Pustular eruptions	antibiotic ointments
Follicular (pyogenic)	antibiotic ointments
Follicular (dry)	vitamin A ointments
Flaired lesions	silver nitrate solutions
Weeping eczemas	astringent wet packs
Urticarial eruptions	antipruritic lotions
Ulcers (pyogenic)	antibiotic ointments
Ulcers (stasis)	stimulating ointments
Excoriations	antipruritic ointments
Purpuric eruptions	mild astringent lotions
Verrucous lesions	keratolytic ointments
Pigmented lesions	Benoquin Ointment
Depigmented lesions	Psoralen lotions or ointments
Atrophic lesions	estrogen ointments or oils

In addition to topical therapy internal treatment usually is required depending on the diagnosis. Specific drugs, antihistamines, antibiotics, sedatives, vitamins etc., may be required in order to correct all the factors that have interfered with the normal metabolism of the skin.

Dressings. Acute dermatoses should be protected from irritation by the clothing. This can be accomplished by using light, thin dressings, tubular gauze (Tubegauze, Scholl) by sewing muslin in the sleeves or the trousers or by the use of white cotton stockings from which the foot has been cut off. Adhesive plaster should not be used in the vicinity of eczematoid or pustular areas. Ointments will not come through dressings if waxed paper is used or if talcum powder is sprinkled over the surface of the medication. Gauze must never be used to dress skin eruptions as it sticks to the secretions permits ointments to seep through and irritates weeping surfaces. Linen or bleached muslin if clean and without wrinkles is preferred.

OCCLUSIVE DRESSINGS (1) prevent trauma to the affected area by clothing scratching and rubbing (2) ensure against spreading of infectious lesions and (3) break up the scratch reflex. This type of dressing is useful in paronychia, furuncles, pyoderma, pemphigus, localized neurodermatitis and factitious dermatoses.

Baths. The proper type of bath depends upon the type and the stage of the eruption and the presence or the absence of itching and constitutional symptoms.

CLEANSING BATHS are shower baths or tub baths using ordinary toilet soap and water. They are contraindicated in acute and subacute dermatoses but are desirable in chronic conditions (acne, seborrheic dermatitis, psoriasis, etc.)

ACID BATHS usually consist of the addition of one half to one pint of vinegar to one half a tubful of warm water. This type of bath is useful in urticaria. The presence of excoriations is a contraindication.

ANTIPRURITIC BATHS are employed in generalized pyrogenic conditions and in disorders characterized by foul-smelling secretions (Darier's disease pemphigus, granuloma inguinale). Boric acid and potassium permanganate are used for this purpose.

Hypertonic salt baths are useful in Ichthyosis and Darier's disease.

COLLOIDAL BATHS which consist of oatmeal, Aveeno and Lint corn starch, are indicated in all acute exudative and pruritic dermatoses, including plant dermatitis, urticaria pityriasis rosea, acute lichen planus etc. The directions for the oatmeal bath are as follows

1. Pour one quart of boiling water over two cups of oatmeal.
2. Cook on the stove until a thick porridge is formed.
3. Fill the bathtub one half full of warm water at 90° F
4. Dissolve one half teaspoon of baking soda in the tub of water
5. Pour the porridge into a cheese-cloth bag and tie securely
6. Swish the porridge bag around in the tub, stirring up the soda until the water is milky white
7. The porridge should be used as a sponge to wipe over the patient and to remove old ointment, lotion or dried scales.

The Aveeno bath requires less time. The patient dissolves 2 cups of the preparation in the bath and bathes in the usual way.

ALKALINE BATHS are sodium-bicarbonate (baking soda) baths. They are useful in all chronic scaly dermatoses when used in conjunction with tincture of green soap.

CONTINUOUS BATHS are used in pemphigus and severe burns.

PHARMACEUTIC PREPARATIONS

Soaps and Cleansers

Indications for the controlled use of soap and water include (1) diseases where grease-solvent action is desired, as in acne acneiform eruptions, seborrhea and seborrheic dermatitis (2) diseases in which a bactericidal effect is important, as in impetigo furunculosis, folliculitis and pyoderma (3) dermatoses characterized by hyperkeratosis, as in lichen pilaris, plantar hyperkeratoses and palmar hyperkeratoses. Darier's disease and psoriasis

and (4) diseases associated with foul secretions, as in hyperhidrosis, bromidrosis and hidradenitis suppurativa.

Contraindications. The use of soap and water should be eliminated or reduced, and soap substitutes prescribed in the following conditions (1) dermatoses associated with extreme dryness, as in ichthyosis, xerodermia phrynoderma, radiodermatitis and occupational "dry skin" (2) eczematous dermatoses, bacterial or mycotic (3) atopic eczema and (4) acute inflammatory dermatitis.

Hard Soaps. Castile (olive oil) is the best mild hard soap for general use. Laundry soaps are very alkaline and are useful in scabies psoriasis and chronic seborrheic dermatitis.

Soft Soaps. Tincture of green soap (olive or linseed oil) which is a good detergent, is ideal for the removal of crusts and is effective in oily seborrhea and acne.

Germicidal soaps contain hexachlorophene (G11) and are indicated in acne, syphilis vulgaris and folliculitis. Unless used at regular intervals they do not reduce the normal bacterial flora to any extent. The following soaps contain G11 Dial (Armour) Septisol (Vestal) Hex-O-San and pHisoex.

Medicated soaps contain benzoin sulfur Ichthyl mercury or tar. Their efficacy is doubtful because inert ingredients may be present.

Superfatted soaps (Basis, Hazeline) are the mildest of soaps. They are indicated in all conditions where an ordinary soap would be irritating (mild eczema, xerodermia, senile pruritus, etc.)

Sulfonated detergents, or "surface-acting agents," are "soapless" nonlathering products which are indicated when sensitivity to ordinary soap is present. Examples are Acidolate (White) Detergol (Doak) Tenuis (Doak) Hand Cleanser (Breck) Terjolate Household Detergent (White) and pHisoderm (Winthrop)

EMULSIONS

Emulsions are combinations of an oil and a lotion and are applied as wet packs on muslin or gauze during the day. There are cream lotions (liniments) which are indicated in acute dermatitis when the skin is too dry to tolerate calamine lotion. They have a soothing protective effect. The various types of subacute eczema respond well to this type of therapy.

Columbus Liniment (N.F.)

Neocalsulfon	80
Zinc oxide	80
Olive oil	
Lime water aa ad	1000

S. Saturate gauze pads and apply to area.

Oils

Mineral oil, castor oil and cotton-seed oil are used to remove crusts, scales, old medications and dried secretions. They are applied on cotton sponges or as compresses and are left to soak in for several hours. Nutritive oils (cod liver oil and olive oil) are used plain or irradiated, in ichthyosis and hypovitaminosis A for their systemic action, as well as for their local action.

Ointments

Ointments (salves, creams) consist of liquid or solid drugs in a greasy base which may be of the inert, the oil-in-water (washable) or the water in-oil types.

Washable-type ointment bases (O/W) are used where prolonged contact is unnecessary on hairy areas and for the more superficial dermatoses. Examples include Multibase Dermabase, Neobase and Almay Emulsion Base.

Water-in-oil bases (W/O) are more penetrating more heating and more stable. They are used in the dry scaly dermatoses, e.g., neurodermatitis, senile pruritus, etc. Examples are Hydro-sorb, Polysorb Qualatum and Velvachol.

Inert ointment bases are used chiefly for lubrication and as a vehicle for drugs used in the treatment of psoriasis. They include white and yellow petroleum and boric acid ointment.

Pastes

Pastes are similar to ointments but are thicker because the dry ingredients comprise 50 per cent of the preparation. They are more protective, more absorbent, more soothing and less heating than ointments but are more difficult to remove and require dressings. Since pastes are drying, they are indicated in subacute dermatoses. Sulfur tar etc may be added when indicated. Pastes should be smeared on pieces of muslin and applied to the affected part.

Lassar's Paste (without salicylic acid)

Zinc oxide		
Corn starch	8A	25.0
Petrolatum alba q.s. ad		100.0

Wet Dressings

Wet dressings are used on edematous erythematous eruptions, weeping dermatoses and all primary and secondary pyogenic infections. The following solutions are employed saturated boric acid, physiologic saline Burow's solution aluminum acetate tannic acid (2 per cent) Alibour solution (1:10) potassium permanganate and magnesium sulfate. A half dozen layers of gauze are used and are kept wet by applying the solution with a syringe at frequent intervals to prevent drying. Plastic mitts or oiled silk must not be used as they prevent evaporation and the cooling effect and, thus, defeat the purpose of the wet pack.

A macerating action (removal of crusts) may be obtained with warm boric acid or 3 per cent sodium bicarbonate solutions a cooling effect (acute dermatitis) with Burow's solution diluted with 20 parts of water a hyperemic action (cellulitis) with hot boric or physiologic saline and a bacteriostatic action (infectious eczematoid dermatitis) with solutions of 1:1,000 silver nitrate solution Zephiran or boric acid.

Lotions

Lotions are liquid preparations containing soluble or insoluble drugs. Bases for lotions depend upon the type of eruption. The following are examples distilled water or rose water witch hazel, camphor water peppermint water lime water from 15 to 70 per cent alcohol dilute solution of lead subacetate. Indications for lotions are erythematous, macular vesicular and acute follicular eruptions. Lotions should be shaken poured into a saucer applied with a brush or a piece of linen and then permitted to dry.

Soothing Lotions.

(1) Basic formula for shake lotion

Bentonite	4
Zinc oxide	
Talc aa	50
Glycerine	2
Water ad	100

M ft bottle

*Astringent Lotions.**N oculumine Lotion*

Prepared neocalamine	15
Magma of Bentonite	40
Water to make	100

Stimulating Lotions. These are used mainly on the scalp to promote the growth of hair in alopecias or to overcome dandruff. For examples see page 497.

Note. Calamine lotions should not be used longer than 3 days in cold weather and should not be used in the aged since the lotions are very drying and chap the skin.

Protectives

Protectives are soothing, cooling preparations which form a film on the skin. They have little or no therapeutic value.

Powders. Zinc oxide zinc stearate bismuth subnitrate

Ointments. Zinc oxide ointment. Silicone ointments.

Paints. Tincture benzoin flexible collodion zinc gelatin.

Lubricants

Lubricants are oily or mucilaginous preparations used in generalized, scaly dermatoses.

Oils. Liquid paraffin, olive oil, castor oil, cotton seed oil. Lubiderm (Abbott) and Liquid Nivea (Duke)

Demulcents. Glycerite of boric acid Wibi lotion Nepto lotion

THE SPECIFIC ACTION OF DRUGS

Anhidrotics. To check excessive sweating Pro-banthine (Searle) or Pamine (Upjohn) may be useful in some cases, but the frequency of side effects limits their use. Aluminum chloride solution (25 per cent) N.F. is useful as a local application on unbroken skin.

Antiseptics. The following drugs are useful antiseptics, but many patients are sensitive to them. Hot boric packs accomplish a great deal in acute pyogenic disorders with less danger.

LOTIONS and SOLUTIONS. Saturated boric solution, potassium permanganate, and tyrothricin.

ONTAINMENTS. Ammoniated mercury yellow oxide of mercury Vioform tyrothricin penicillin Bacitracin Aureomycin Terramycin neomycin, streptomycin polymyxin phenylmercuric nitrate bismuth tribromophenate (Veroform) boric acid Quinolol (oxy quinoline sulfate)

DYES are used only on covered areas since they stain and are not removed easily. The chief dyes employed in dermatology are the following

Castellani's paint used in chronic seborrheic dermatitis, tinea cruris and chronic dermatophytosis of the feet.

Gentian violet (2% aqueous) used in moniliasis (oral vaginal or intertriginous) and as a bacteriostatic agent in nonspecific ulcers.

Brilliant green used in 1 per cent concentration with 6 per cent salicylic acid and 12 per cent benzoic acid in infected chronic dermatophytosis of the feet.

Irritants are usually used in the treatment of alopecia. They act by increasing the local blood supply. The irritants used commonly include phenol, tricresol, capsicum catharides, tincture of iodine, formic acid and alcohol.

Parasiticides should be used with care. They may produce contact dermatitis if used in strong concentrations or if applied to sensitive areas.

ANIMAL PARASITICIDES phenolated camphor DDT benzyl benzoate Eurax (Geigy) Kwell (Commercial Solvents) Cuprex (Merck) Bormate (Wyeth) Gamiso (Texas) and Hexacide (Central)

VEGETABLE PARASITICIDES Sulfur salicylic acid and benzoic acid, ammoniated mercury Anthralin iodine, thymol, carbolfuchsin, gentian violet, sodium thiosulfate, sodium, copper and zinc salts of propionic and undecylenic acids salicylanilide Verdefam Vioform and Diodoquin

Drugs used for systemic fungus infections include the iodides (sporotrichosis) penicillin (actinomycosis) Terramycin (actinomycosis) stilbamidine (blastomycosis) diethylstilbestrol (blastomycosis) and Diodoquin and nystatin (moniliasis)

Reducing agents abstract oxygen from the tissues and thus hasten keratinization. They are valuable in the treatment of psoriasis and chronic eczema. Important ones are Anthralin, tar pyrocatkol, mercury sulfur Ichthyol (Ichthammol)

Caustics are used to destroy epithelial proliferation. They should not be used in the treatment of moles or precancerous dermatoses because of the danger of stimulating malignant degeneration.

MILD CAUSTICS Chromic glacial monochloroacetic, dichloroacetic, and trichloroacetic acids, benzoic acid and salicylic acid resorcinol (20 per cent) silver nitrate (10 per cent) potassium hydroxide (25 per cent)

STRONG CAUSTICS Nitric acid, phenol, acid nitrate of mercury formaldehyde solid carbon dioxide liquid oxygen and nitrogen.

Anti-inflammatory Ointments. These corticosteroids for topical use are effective in many cases of atopic eczema, small areas of seborrheic, infantile and contact dermatitis and idiopathic pruritus vulvae et ani. The tablets of hydrocortisone may be used in a suitable base to meet the requirements of the clinical lesion or the commercial ointments may be prescribed. In some cases, the effect is only temporary. Reliance must not be made on these ointments alone but other necessary measures may be indicated.

The following products are available. Cortidome Ointment (Dome) Hydrocortisone Ointment (1.25%) Cotril Topical with 3 per cent Terramycin (Pfizer) Neocortel Ointment with 1 per cent Neomycin (Upjohn) Meticorten (Schering) and fluorhydrocortisone ointment.

SYSTEMIC THERAPY

Bleaching Agents. Benzoquin ointment (Elder) is useful in reducing superficial melanin pigmentation. Chloasma but not pigmented moles may respond to daily applications for several weeks. If irritation develops, the ointment should be diluted with hydrophilic base and reapplied after the contact dermatitis has cleared. Mercury creams should not be used concurrently as an unsightly pigmentation may result.

Epithelial stimulants are used in the treatment of ulcers and include balsam Peru allantoin aloe vera, radon ointment, chlorophyll Ichthyol and scarlet red.

Epidermal stimulants are used in chronic eczemas to promote healthy epidermal cells. Ichthyol is the least irritating of the group which includes naltalan, oil of cadberry oil of cade, crude coal tar liquor carbonic detergents pix liquida and liquor picis carbonia.

Nonspecific desensitising agents include

Calcium gluconate.

Autochemotherapy

Intravenous-triple typhoid vaccine.

ACTH

Prednisolone

Arthur Gel.

Meticorten.

Cortisone

Hydrocortisone

Prednisolone

Sex Hormones.

Estrogens are useful in dermatoses which are associated with artificial or normal menopause (senile vulvitis pruritus vulvae,

chronic neurodermatitis nail dystrophies, actinic eruptions, kraurosis vulvae etc.) We have seen no benefits in menopausal alopecia. Estrogens may improve resistant female acne but the dosage must be individualized given in the last half of the menstrual cycle and side reactions must be avoided. Administration is by oral route (Estradiol Tablet U.S.P. 0.2 mg. Theeol or Premarin) or by injection (Estrone Injection U.S.P. 1 mg. or Hexesterol).

ANDROGENS may be effective in males with senile pruritus, mycosis fungoides, resistant types of chronic lupus erythematosus, apocrine dermatoses, to control the edema from corticosteroid administration and in early cases of leukoplakia. The hormone is given as Testosterone Propionate Injection U.S.P. (25 mg. intra muscular).

Antihistaminics have a suppressive but not a curative action in urticaria, angioneurotic edema and some drug eruptions. Their sedative effect is valuable in dermatoses associated with pruritus, neurodermatitis and autosensitization with the production of Ika. These drugs may have no effect in some cases and occasionally may even produce drug eruptions.

Sedatives are used in acute disseminated neurodermatitis, atopic eczema and pruritus vulvae in disturbed patients. The following drugs may be used benedryl barbiturates, chloral, reserpine and chlorpromazine.

Vasodilators are used in peripheral vascular disease and early cases of progressive scleroderma. These drugs include nicotinic acid amide Priscol, Etamon Chloride (Park Davis) aminophyllin acetylcholine and sodium nitrite.

Anticholinergic drugs which have a parasympatholytic action are useful in cholinergic urticaria functional hyperhidrosis, etc. These drugs include Prantal (Schering) Pro-Banthine antihistaminics and belladonna derivatives.

Heavy metal antidotes neutralize the toxic effects on the tissues in arsenical gold bismuth and mercury intoxications. BAL (British anti Lewisite) is at present the most efficient drug for this purpose.

Hyaluronidase is an enzyme capable of hydrolyzing or depolymerizing hyaluronic acid a mucopolysaccharide which acts to hold cells together in a jellylike matrix. The enzyme is injected intracutaneously in small lesions or introduced by iontophoresis in more extensive conditions.

INDICATIONS early leiods localized myxedema morphea and generalized scleroderma (iontophoresis)

Sclerosing agents are used in the treatment of varicose veins, raised angiomas and in some sebaceous cysts.

Hypnotism may be useful in some cases of disseminated warts and selected cases of intractable pruritus.

Hemostatic drugs are used in purpuric eruptions. These include snake venom solution (moccasin) rutin ascorbic acid and vitamin K.

Vitamins are used for their specific or pharmacodynamic effect in nutritional dermatoses, septic eczemas and, in a few cases for their specific effect.

Vitamin A may be useful in follicular acne pityriasis rubra pilaris, Darier's disease phrynoderma, xeroderma some cases of hyperkeratosis of the palms and the soles and onychorrhexis. Injections are preferable to oral administration. The vitamin has a restraining effect upon the keratinization functions of the epithelial cells. It is administered as Oleovitamin A Capsules U.S.P., Afarin (Winthrop) or preferably as the water soluble Aquasol A (Funk)

Vitamin B₁ (thiamin) Its value in herpes zoster and herpes simplex is doubtful.

Vitamin B₂ (niacin) is important in the treatment of pellagra. Riboflavin is used in glossitis and seborrhealike eruptions of ariboflavinosis.

Vitamin B₁₂ may be useful in seborrheic dermatitis as supplemental therapy. The dose is 10 to 15 mcg 3 times a week by intramuscular injection.

Vitamin C is indicated in scurvy purpuric eruptions, gingival lesions, indolent ulcers, onycholysis acrocyanosis and chloasma.

Vitamin D₂ (Calciferol U.S.P) is used in lupus vulgaris acrofuloderma and other tuberculous dermatoses and in chronic types of parapsoriasis.

Vitamin E has been employed empirically in the collagen diseases.

Vitamin K is used in chronic urticarias and purpuras when the prothrombin level of the blood is reduced. It is prescribed as Menadiolone U.S.P. Hyklone (Abbott) or Synkamin (Parke Davis)

Nuclear toxins are used in the lymphoblastomas to reduce the multiplication of abnormal cells and to increase the duration

of the remissions. Individual response and toxic reactions vary. These drugs include urethane P_{12} , nitrogen mustards and triethylene melamine (TEM).

Drugs with a nonspecific action but which are valuable in certain dermatoses include arsenic (dermatitis herpetiformis, atopic eczema and lichen planus), sulfapyridine (dermatitis herpetiformis), Aminopterin (acute leukemia), Aralen and Camoquin (chronic lupus erythematosus), bismuth (lichen planus and flat juvenile warts), mercury (flat juvenile warts and lichen planus), thyroid extract (acne vulgaris and psoriasis) and iodides in late syphilis and the deep mycoses.

Aminoacids are used as supplemental therapy in conditions with nitrogen loss as in pemphigus, exfoliative dermatitis, nutritional eczema and bed sores.

Vasoconstrictor drugs are employed chiefly in urticaria and angioneurotic edema. Adrenalin is used for its rapid effect and adrenalin in oil or ephedrine sulfate for a more prolonged action.

Adrenocorticotrophic Hormones. ACTH, cortisone, hydrocortisone, prednisone and prednisolone inhibit or reduce acute inflammatory reactions in the skin resulting from various known and unknown causes. They produce remissions rather than cures in acute disseminated lupus erythematosus, pemphigus, dermatomyositis, early scleroderma, periarteritis nodosa, severe drug eruptions, exfoliative dermatitis and psoriatic arthropathy. Since these drugs may produce serious side reactions, close observation is necessary during the period in which they are used.

Placebos are used sometimes in dermatoses of emotional origin, in overtreatment dermatitis and in cases in which laboratory data is being awaited in order to make a diagnosis. Boric acid in ointment or 4 per cent solution is the commonly used placebo. It holds the patient's confidence and gives the dermatologist time to make the required investigations.*

PHYSICAL METHODS

CRYOTHERAPY (COLD)

Solid Carbon Dioxide. When carbon-dioxide gas is released under pressure at room temperature it is changed into a solid state. The snow thus produced is compressed into a pencil point, an ear speculum, a wood block or any of the more expensive

Abramowitz, E. W. Use of placebos in the local therapy of skin diseases, *New York State J. Med.* 48: 1927-1948.

outfits sold commercially as a mold. The preparation is employed as a caustic and is moderately destructive in its action. Slight moderate or deep pressure is used. One minute is usually sufficient for each application. Unless applied very superficially scarring is apt to result.

INDICATIONS. Angiomas, chronic lupus erythematosus, keratosis and creeping eruption.

Carbon-dioxide slush which is made by adding acetone to the snow is applied with a brush to the area to be treated. Its chief value is in the therapy of acne scarring.

Liquid Oxygen or Nitrogen. More destructive than solid carbon dioxide their action is more rapid. The material is kept in a vacuum bottle and is applied to the lesion with a cotton swab. Indications include resistant plantar warts, hemangiomas, keratosis, leukoplakia and folliculitis keloidalis.

ABRASIVE THERAPY

Acne scars and variola and varicella scars and superficial tattoos can be made less noticeable by sandpaper abrasion or by the use of a motor driven wire brush using Fluoro-ethyl or Frigiderm local anesthesia.*

ELECTROTHERAPY

Electrolysis is used in the treatment of superfluous hair, adenoma sebaceum, telangiectasis, colloid milium, small verrucae and spider nevi. For electrolysis of the hair follicles, the patient grasps a metal electrode or a pad soaked in saline solution. This is connected to the positive pole of a galvanic battery. The negative pole is connected by means of a suitable holder to a flexible platinum needle having a stub point. This needle is inserted gently into the hair follicle until it reaches its base. Care should be taken that the current is not passing when the needle is inserted or withdrawn. A current of from $\frac{1}{2}$ to 2 milliamperes is allowed to pass until bubbles appear at the mouth of the follicle. This occurs after about 30 seconds when it is found that the hair may be withdrawn quite easily. If the hair is not loose the needle must be reinserted and further current must be passed. From 30 to 40 hairs are removed at a sitting. The operation,

*Kurtin, Abner. Correctin plaqueing (the skin), A.M.A. Arch. Dermat. & Syph. 68: 389, 1912 and Emerson, F. C. Further developments in the treatment (the skin) by surgical abrasion. Plast. & Reconstruct. Surg. 12: 1, 1953.

which is moderately painful, produces a temporary small edematous macule, leaving a very faint scar. A recurrence of 25 per cent or more hairs is to be expected with this method.

Pulverization is the superficial application of a high frequency current, resulting in a stream of fine sparks which flow into the tissues from the point of the metal electrode. Although the method is painful only a few seconds are required for blanching. Healing requires from 10 to 14 days.

Electrodesiccation is a form of contact surgical diathermy in which the needle is inserted on, or just within the lesion to be destroyed.

In electrocoagulation, the bipolar method is used the current passing from electrode to needle with destruction of the tissue between the poles. Local anesthesia is employed, the desiccated tissue is curetted to prevent infection and a sterile dressing is applied.

INDICATIONS: Carcinoma, leukoplakia, verrucae, nevi, xanthoma and keratoses.

Surgical diathermy is the production of heat within the tissues with the endotherm knife resulting in coagulation of tissues. Experience is required to determine the amount of current necessary to cut through the tissues so that primary healing may occur.

INDICATIONS: Leukoplakia, nevi, carcinoma and intra-oral growths.

Electrocautery. The actual cautery is the best method to destroy structures which bleed easily and to stop bleeding in minor surgery. Scars may result which are often hypertrophic. Using a cherry red heat the lesion can be destroyed under local anesthesia by using rapid and repeated applications of the point.

INDICATIONS: Warts, granuloma pyogenicum and keratoses.

THERMOTHERAPY

Infra red radiation is a form of radiant heat with wave lengths beyond the visible range of the spectrum. The biologic effects are vasodilatation (active hyperemia) and moderate analgesia.

INDICATIONS: For the relief of pain in carbuncles and cellulitis, to stimulate sluggish ulcers and to improve the local circulation in peripheral vascular disease.

IONTOPHORESIS

This is the introduction of the ions of various metals (zinc, copper magnesium etc.) and hyaluronidase into the affected tissues by means of the electric current.

INDICATIONS. The method has been used in treating warts, scleroderma, fungus infections, etc., with varying results.

PHOTOTHERAPY (LIGHT)

Ultraviolet rays are obtained from the sun carbon-arc lamps and mercury vapor lamps. The far-ultraviolet rays 2,000 to 3,000 Å, are invisible. They have a low penetration (1 mm.) and a biologic and chemical action. Their effect on living tissue depends upon the amount absorbed which in turn depends upon their intensity reflection, penetration and freedom from obstruction.

The biologic action of ultraviolet rays consists of (a) erythema and (b) systemic effects. The erythema occurs from 6 to 12 hours after exposure. First second- third- and fourth-degree burns may result, depending upon the degree of exposure. The systemic effects consist of relaxation, induction of sleep increased physiologic activity of the skin (temporary rise in calcium and vitamin D) and an analgesic action on the cutaneous nerves, resulting in temporary relief from itching.

Overexposure to the rays, or decreased intervals between treatments results in burns. Freckles and tanning usually follow repeated exposures in certain individuals. Sensitivity to the rays, resulting in some cases from a disordered hematoporphyrin metabolism, may play an important role in hydroa vacciniforme xeroderma pigmentosum chronic actinic dermatitis and keratoses.

INDICATIONS for air-cooled ultraviolet light therapy: acne vulgaris (acute cases juvenile acne pitted scars recurrences when x-rays have failed) furunculosis, erysipelas pyoderma in infants, psoriasis (tanning must result to produce involution) pityriasis rosea, seborrheic alopecia, alopecia areata ichthyosis (temporary improvement)

CONTRAINDICATIONS include acute dermatitis lupus erythematosus herpes simplex pellagra "sensitive skin" sycois barbae acute psoriasis and acute seborrheic dermatitis.

THE WATER-COOLED MERCURY VAPOR LAMP is a small lamp with a quartz window the heat rays of which are absorbed by circulating water in a jacket around the burner

Indications for water-cooled ultraviolet therapy include. lupus vulgaris, tuberculosis verrucosa cutis (general ultraviolet baths also) and port wine angioma.

X ray therapy is an important component of dermatologic therapeutics. Before acquiring an x ray machine one should consider the following factors flexibility of tube head ease of manipulation cost and availability and promptness of service. The x rays are invisible electromagnetic waves produced by a bombardment of electrons from the negative cathode against a target, or anode. The dose or quantity of radiation is measured clinically by the erythema or epilating dose. A more accurate dosage has been established in the standard international unit or roentgen (r) A roentgen is the quantity of roentgen radiation which in 1 cc. of air at 0 C. and 76 mm. mercury produces such a degree of conductivity that 1 electrostatic unit of charge is measured at saturated current. One skin unit is about 325 r

X-rays are used filtered for deep penetration and unfiltered for superficial penetration They may be administered fractionally subfractionally or intensively (full erythema dose)

CONTACT X RAY THERAPY is given with special machines which deliver large doses (from 10,000 to 16 000 r per minute) through a small portal (2.5 cm.) Because of the rapidity with which large doses can be given this type of therapy is advantageous in treating small carcinomata eyelid tumors and verrucae

PRECAUTIONS are necessary when employing x-rays in treatment. Irritating applications should not be permitted for at least one week following therapy Treatment should be withheld also in cases which have received recent radiation of unknown dosage Other contraindications are severe blood dyscrasias, chronic tuberculosis and atrophic skin disorders.

X RAY APPARATUS is complicated and the dosage must be checked bi-annually by a physicist to assure the proper measurement of radiation Shock proof apparatus is available, consisting of automatic timers interrupted transformers and Coolidge tubes, with all exposed parts protected by metal shielding and insulated cables.

The beneficial effects of x ray radiation in the treatment of skin diseases result from the following actions (1) inhibition of glandular hyperactivity and secretions, (2) reduction of the rate or cessation of cell division (3) reduction of the sensitivity of the nerve endings, (4) modification of the soil, inhibiting

bacterial or mycotic growth and (5) desensitization of epidermal and dermal tissue.

Burns may occur from (1) improper dosage, (2) improper protection of normal tissue cross-firing back-scattering and overlapping of the rays (3) repeated treatment of recurrences, (4) failure to obtain a history of previous radiation therapy (5) lack of experience, (6) lack of plan (7) use of diagnostic machines to give superficial therapy without changing the setup.

INDICATIONS for x-ray therapy include acute chronic eczema, localized neurodermatitis, lichen planus sycois vulgaris and paronychia, persistent hyperhidrosis furunculosis paronychia, warts, keloid, carcinoma and mycosis fungoides.

Radium is similar to the x-rays in its biologic action. It can be concentrated into small needles tubes or plaques so that small lesions can be treated intensively. By changing the position of the radium cross-firing of the rays can be obtained, with exposure of all parts of the lesion. Interstitial radium is used for recurrent persistent subcutaneous lesions deep infiltrations, and especially tongue cancers. Radium is dangerous in the hands of the inexperienced.

RADON BATHS containing the emanations, are useful in treating carcinoma. The dosage of radium depends upon the area, the thickness and the type of growth.

INDICATIONS FOR RADICAL. Malignancies angiomata keloids and warts.

GENERAL THERAPEUTIC SUGGESTIONS

Do not prescribe a proprietary ointment or lotion unless you know the composition and the strength of the ingredients.

Do not prescribe strong medication for the tender areas, including the flexure surfaces of the extremities the face, the neck, the mucocutaneous areas the genitals and the dorsum of the hands and the feet.

When the patient has an extensive or severe eruption it is important to see him daily in order to determine the therapeutic response.

Do not fail to hospitalize patients or seek consultation for rapidly spreading eruptions.

Do not use or permit the patient to use U.S.P. ammoniated-mercury sulfonamide or sulfur ointments, coal oil, turpentine,

rubbing alcohol tincture of green soap tincture of iodine or mercurial preparations on vesicular or eczematoid eruptions.

Do not permit adhesive plaster to be used in the vicinity of an eczematoid or a pustular eruption even if there is no history of sensitivity to it.

Do not use alcoholic lotions on excoriated areas.

Do not permit yourself to overstep the limits of safety in treating a refractory condition when employing potent drugs or x-ray therapy.

Do not adopt a pessimistic attitude toward the patient with psoriasis.

Do not use penicillin locally or parenterally for minor dermatoses because of the danger of sensitization.

Do not forget to think of pemphigus when a bullous eruption in the mouth fails to respond to local or vitamin therapy.

Do not fail to think of a factitious element when a lesion or an eruption appears to spread or to recur under proper therapy.

Do not apply patch tests during the active phase of an eczema or a dermatitis.

Do not write a complicated prescription when a simple one will do.

Do not apply ointments or pastes to weeping areas, as they tend to seal the exudate. This promotes bacterial contamination.

Do not fail to get a pathologic report on all suspicious benign tumors, as well as on clinically malignant growths.

Do not use Whitfield's ointment, full strength, on any areas except the palms and the soles of adults.

Do not fail to reduce the strength of drugs used in ointments, from one tenth to one third in infants and children.

Do not fail to investigate for external irritants when a simple eruption fails to clear up under ordinary routine therapy.

Do not prescribe tar ointments for hairy areas because of the danger of folliculitis.

Do not forget to examine the scalp in all dermatoses of the face and the trunk. Seborrheic dermatitis, like syphilis, is a great imitator.

Do not prescribe strong medication for dermatoses about the eyes without warning the patient about the possible danger of an edema of the eyelids.

Do not depend upon ultraviolet radiation as a 'cure-all.'

Do not use colored dyes on skin eruptions or lesions and then expect the consultant to make a diagnosis.

Do not use calamine lotion on hairy or weeping areas. It will cake and cause irritation. If used on a dry or senile skin for longer than 24 hours, it will cause chapping.

Do not use gauze next to an irritated skin. Bleached muslin or linen is preferred.

Do not treat potentially malignant lesions with the "electric needle."

Do not prescribe strict diets for patients with skin eruptions unless there is a definite indication and a definite diagnosis has been made.

Do not discontinue a local application or a plan of treatment so long as it is yielding benefit.

Do not use the sulfonamide drugs with their toxic potentialities orally if the condition is amenable to local therapy.

Do not give antisyphilitic therapy to any patient unless the diagnosis of syphilis has been verified by a reliable history, a physical examination, a darkfield examination, a blood test or a biopsy.

Do not use oral penicillin therapy in the treatment of syphilis.

Do not use local sulfonamide therapy in skin conditions because of the danger of local and general sensitization.

Do not use adrenocorticotrophic hormones orally or topically unless there is a definite indication for its use.

Do not let your interest in the disease eclipse your interest in the patient.

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Eruptions of the Hands

ACUTE DERMATITIS DUE TO
PRIMARY IRRITANTS
ALLERGIC CONTACT DERMATITIS
INFECTIOUS ECZEMATOID
DERMATITIS
DYSHIDROSIS
NUMMULAR ECZEMA

POMPHOLYX
DERMATOPHYTOSIS
DERMATOPHYTIDS
PUSTULAR BACTERIDS
PSORIASIS
PUSTULAR PSORIASIS
FOOD SENSITIZATION ERUPTIONS

ALMOST any dermatitis can involve the hands and the fingers at one time or other but the erythematousquamous and the vesicopustular eruptions cause the most difficulty in diagnosis. The accuracy of the diagnosis will depend on a thorough history the clinical experience and the open-mindedness of the examiner and an evaluation of all the contributory factors. Confusion exists regarding the cause and the diagnostic criteria of some of these diseases but it seems possible that individual predisposition and multiple factors play an important part in the cutaneous pattern. Cornia* stresses the importance of assessing the various factors that cause dermatitis of the hands.

Types of Eruptions. The clinical appearance of many of these eruptions is often atypical because of secondary infection or eczematization from various causes however the following morphologic grouping seems to be reasonable for diagnostic purposes

- 1 Acute eczematoid dermatitis—primary contact dermatitis and allergic contact dermatitis.
- 2 Vesicular and bullous eruptions—pompholyx, dyshidrosis and drug eruptions.
- 3 Vesicopustular eruptions—bacterids and dermatitis repens.
- 4 Chronic erythematousquamous eruptions—psoriasis, chronic neurodermatitis, chronic senile eczema.
- 5 Chronic patchy eczematoid eruptions—nummular atopic or food eczemas, dermatophytosis.

Cornia, F. E. Eczema of the hands, Canadian M. A. J. 66 451 1952

6 Hypertrophic scaly lesions—hyperkeratotic psoriasis, late syphilis, psoriasis, dermatophytosis and neurodermatitis.

General Diagnosis.* Before a diagnosis is made the entire skin surface should be examined for evidence of psoriasis, a focus of fungus infection, presence of contact dermatitis or atopic eczema. If the eruption is unilateral, a local cause is suggested (fungus or microbe). If the eruption comes in cycles and is associated with a psychosomatic factor the eruption is possibly a neurodermatitis or a pompholyx. If the eruption follows a flare-up of a definite focus of infection a bacterid is a possible diagnosis.

In any event, routine cultures should be made for fungi and bacteria, but their presence may be of no significance. In acute cases it is important to evaluate the factor of occupational contact.

Most cases of contact dermatitis in housewives begin under the wedding ring and spread from there. These cases are common during the winter season. Although patch tests with soaps and detergents are usually unsatisfactory these products often will aggravate a dermatitis of the hand and interfere with proper diagnosis.†

The author uses the following questionnaire which is filled out by the patient. The answers may be helpful in arriving at a working diagnosis.

Questionnaire on Dermatoses of the Hands

Name	Age	M	S	W	D
Address	Zone				
Location of eruption					
Duration					
Where did eruption first begin? Exact site					
Have you had previous attacks?			When?		
What helped it?					
Did the rash start as blister red spot, itch or weeping area?					
What did you use on it first?			What happened?		
Did you have rash on your feet at the time the rash appeared on your hands?					
Do you use rubber gloves when you wash dishes? Do the laundry? Clean the sink or polish silver?					

For a general review of the problem, see Lane, C. G. et al. Dermatoses of the hands, J A M A 128:1917-1945.

†For general discussion of the subject, see Sulzberger M. B. and Baer R. L. Year Book of Dermatology and Syphilology Eczematous Eruptions of the Hands, pp 7-44 1948.

What kind of soap do you use for bath?

Dishes?

Laundry?

Do you have a skin rash anywhere else at this time?

Is your skin usually dry, moist or normal?

Are you taking any medicine internally at this time?

What was the last application you used on your hands?

Have you ever had eczema before?

Where?

Do you consider yourself nervous?

Is the itching worse when you are upset or under a strain?

Do you have a feeling that some particular food you are eating may be the cause?

Do you do gardening? Painting, photography etc. as a hobby?

If employed outside the home what kind of work do you do?

What materials do you handle?

What kind of bag do you carry? Gloves? etc.

Do you shampoo your own hair? What do you use?

Do you have any bad teeth, tonsils, stanzas or frequent sore throat?

What do you think is the cause of your eruption?

Physical Examination. It is important to examine the entire skin surface seeking clues as to the nature of the eruption also the feet for evidence of active fungus infection. A symmetrical eruption on the palms suggests an internal causation while eruptions on the dorsal surface may indicate an external cause. The exact localization of the eruption should be noted—palms dorsum, interdigital spaces, nail fold thenar or hypothenar involvement and wrists.

General Etiology While the cause of many of these disorders is unknown or difficult to evaluate in certain cases, the following factors predispose to dermatoses of the hands

- 1 Dry skin (xeroderma)—contact dermatitis nummular eczema.
- 2 Primary neurovascular instability—dyshidrosis menstrual eczema and atopic eczema.
- 3 Psychosomatic influences—chronic neurodermatitis, pompholyx.
- 4 Excessive sweating—dyshidrosis, fungus infections.
- 5 Excessive exposure to water—Moniliais.
- 6 Infected or irritated fungus infection of feet—dermatophytids.

- 7 Specific allergy to foods or drugs—various types of eczematous or bullous eruptions.
8. Individual predisposition—heredity and “soil” important.
- 9 Menopause may be a factor in some cases of chronic neurodermatitis.

Complications of Hand Eczema. Regardless of the primary cause the following may affect the clinical course and prognosis adversely: Irritation from soaps and washing powders and detergents; secondary contact dermatitis from local therapy; secondary infection; the development of autosensitization; secondary flare-ups from psychogenic causes, menses and systemic antibiotics; secondary food allergy.

ACUTE DERMATITIS DUE TO PRIMARY IRRITANTS

This type of eruption is usually occupational and consists of acute edema, diffuse erythema and vesiculation resulting in weeping and crusting. Symptoms are tenderness and pain and more or less disablement. Complications include secondary infection, autosensitization or eczematids.

Localization. The dorsal surface of the hand and the fingers and the interdigital spaces are the usual sites. The palms are spared because of the protective thickened epidermis.

Etiology. These eruptions usually occur from occupational exposure to primary irritants including acids, alkalis, salts of the heavy metals and fat solvents.

Diagnosis is made by the history (contact with irritant), lack of adequate protection and clinical appearance of the eruption.

Prognosis is good if co-operation is obtained with cure usually within 2 weeks.

Prophylaxis after cure may be obtained by the use of the silicone ointments.

Treatment consists of avoidance of the cause, wet packs of potassium permanganate (1 to 8,000), saturated boric or aluminum acetate (5 per cent) followed by drying lotions and later Lassar's paste. In severe cases ACTH or hydrocortisone shortens the acute phase.

ALLERGIC CONTACT DERMATITIS

This type of eruption consists of ill-defined superficial patches of dermatitis appearing at the site of maximum exposure and usually spreading to involve a larger area. Erythematous in the

beginning the lesions become papulovesicular and later may become eczematoid. Itching is moderate. In housewives, the eruption may appear first under the wedding ring if due to soaps or detergents.

Localization. The eruption may be unilateral or may affect both hands, the dorsal surface of the hand or the fingers or the base of the thumbs or the knuckles. The palms never are involved.

Etiology. Most of the cases in housewives are caused by hypersensitiveness to soaps, washing powders or detergents. These occur during the winter months. Young mothers with young children are predisposed. Any of the contactants mentioned on page 90 may produce the eruption in susceptible individuals. Physicians should guard against secondary contact dermatitis from therapeutic applications.*

Prognosis is guarded, since polysensitivity is common and grease reactions are not unusual.

Treatment. Cotton-lined rubber gloves must be used in the management of household activities. Previous therapy must be discontinued and a fresh start made. Use 1 per cent aluminum acetate wet packs for the acute cases, calamine liniment for the subacute cases and Lassar's paste with or without 3 per cent Aeroform or 5 per cent naftalan or 1 to 2 per cent hydrocortisone ointment in the dry stages. Antihistaminic and antibacterial ointments may cause sensitization. A superfatted soap or one of the "soapless detergents" may be used with more or less safety. Itching should be controlled with one of the oral antihistamines rather than by adding phenol to the local applications. Over treatment must be avoided.

In order to obtain the patient's co-operation the following instruction sheet is given:

- 1 Do not wear rings if your fingers are affected
- 2 Wear cotton gloves while doing household tasks that do not involve the use of water
- 3 Wear cotton gloves covered with rubber gloves (Bluettes) when putting hands in water. Seal off the tops with rubber bands if necessary
- 4 Apply medications according to instructions. Wear thin cotton gloves over salve

*Gaul, L. E. and Underwood, G. B. Dermatitis of the hands resulting from overtreatment, *Am Pract & Dured Treat* 3:186, 1952

5. In washing hands, use Aveeno oatmeal powder or special soap and use lukewarm water *not hot water*. Dry hands thoroughly after washing by patting gently. If using Aveeno sprinkle 1 or 2 tsp. into the hands and add a little water. Rub into a paste. Then apply and cleanse as you would with soap.

6. During cold weather wear warm mittens and put them on before going out of doors.

7. Do not remove medications unless for special reason. Put new applications right on top of old. If removal is necessary use oil or Allercreme to loosen medication.

8. Do not manicure your nails while your eruption is active.

9. Do not shampoo hair, do any painting or use detergents or Chlorox until the hands are completely well.

10. Rest and quiet are important factors in helping the skin heal.

11. When peeling onions, garlic, tomatoes or oranges wear cotton-lined rubber gloves to avoid irritation.

12. Keep fingers away from the nose. Infections start that way.

13. If hands get worse between office treatments (1) make a list of everything you handled, touched or used on your hands or fingers 12 hours before the flare-up began and (2) list any mental strain or worry that might have had an adverse effect on the eruption.

14. Do not give any home permanents to yourself or anyone else.

INFECTIOUS ECZEMATOID DERMATITIS

This is an acute, subacute or chronic patchy eruption usually affecting the fingers, and is characterized by vesicopustules, folliculitis or crusted eczematoid lesions. Itching is variable. Spreading is by peripheral extension.

Localization. The fingers usually are affected. When caused by the staphylococcus the hairy parts are affected first. Any part of the hand may be involved. Some cases start in a paronychia, while many cases are secondary.

Etiology. The cause is a low-grade sensitization to the staphylococcus or the streptococcus or an alteration in the host-bacteria relationship. In some cases the eruption appears secondary to a pre-existing dermatosis.

Diagnosis is made by a process of elimination: clinical appearance, bacteriologic cultures and response to therapy.

Prognosis is good in most cases if sensitization does not occur to local antibacterial medication.

Treatment consists of hot boric packs in the acute cases, and Neocortef, bacitracin or neomycin ointments in the chronic cases. X-ray therapy, desensitization with a series of autogenous or stock vaccines or a broad spectrum antibiotic (avoid penicillin) may be useful in refractory cases. Focal infections in the nose and the throat or the vaginal tract should be treated to avoid contamination of the fingers.

DYSHIDROSIS

This is a recurrent vesicular eruption, a form of miliaria, which affects the fingers and the palms and is associated with hyperhidrosis and nervous tension.

The onset is sudden, and after several days of moderate itching discrete, noninflammatory tiny vesicles appear on the sides of the fingers or the palms. These do not rupture but dry up spontaneously leaving a collarlike thin scale.

Localization. The palms, the sides of fingers and occasionally the flexor surfaces of the wrists are the usual sites.

Etiology. Individual predisposition and an abnormal response to the composition of the sweat are possible factors. Shelley considers dyshidrosis a vesicular epidermal reaction pattern and not primarily a sweat retention disorder.

Prognosis. Recurrences are common, secondary complications are rare and the response to therapy is good.

Treatment. Exertion and local irritants should be avoided. Mild sedation may be necessary. Astringent packs (1 to 8,000 potassium permanganate) followed by 5 per cent salicylic acid in alcohol applied three times daily are usually successful.

NUMMULAR ECZEMA

This is a patchy type of eczema consisting of one or more discrete coin shaped sharply demarcated lesions. At first the patches are papulovesicular and later become erythematous plaques with central clearing. Itching is mild to severe.

Localization. Any part of the dorsal surfaces of the hands and the fingers may be involved. In some cases the forearms and the legs also may be sites.

Differential diagnosis is chiefly from contact dermatitis.



FIG. 210. Pompholyx (From Dr Roy L. Kile)

Etiology The disease is associated with a dry skin and is aggravated by alkaline soap during cold weather. Nervous tension may be a link in the chain of causes.

Prognosis is good but recurrences are common every winter.

Treatment consists of astringent wet packs in the acute cases, Vioform cream or hydrocortisone ointments in the subacute cases and x-ray therapy with mild tar ointments in the chronic type. Mild sedatives and large doses of vitamin A appear to shorten the course of the attack.

POMPHOLYX

While the term is a general one it should be limited to those acute eruptions of the palms and the soles which appear suddenly as a result of an acute psychosomatic episode.

The eruption consists of deep-seated noninflammatory vesicles, grouped or closely aggregated with normal intervening skin. Itching or burning precedes the appearance of the lesions.

Localization. The palms, the palmar surfaces and the sides of the fingers and the wrists usually are involved.

Etiology Individual predisposition and neurovascular instability are important factors, and sudden emotional disturbances may precipitate the eruption. Choline may be the sensitizing agent.

Prognosis is good, but recurrences are common.

Differential diagnosis must be made from vesicular "ids" resulting from dermatophytosis of the feet or contact dermatitis with autosensitization*.

Treatment consists of 5 per cent salicylic acid in calamine lotion followed by soothing ointments when desquamation begins. Sedation anticholinergic drugs—Antrenyl (Ciba) Bardase (Parke-Davis) Co-Elorine (Lilly) Pro-Banthine (Searle) or Prantal (Schering)—and superficial x ray therapy are often helpful.

DERMATOPHYTOSIS

Fungus infections limited to the hands are not common because local conditions are not satisfactory for their growth. The lesions may be secondary to nail involvement or they may be associated with dermatophytosis elsewhere. Physicians should avoid calling dermatoses of the hands a form of "athlete's foot" where adequate proof is lacking.

The usual type is a chronic squamous lesion studded with vesicles at the periphery and extending slowly. If the palm is involved the lesion is usually hyperkeratotic or lichenified.

Localization. Any part of the hand may be affected. In monilial infections, the interdigital spaces and the nail folds may be involved also. Trichophyton infections are almost always unilateral.

Etiology Those individuals whose occupation necessitates constant immersion of the hands in water are predisposed. *T. rubrum* is a frequent invader.

Diagnosis is based on positive cultural or microscopic findings for fungi and not on clinical appearance. The presence of active foci elsewhere may be suggestive but not diagnostic.

*The details of differential diagnosis are discussed by Becker S. R. "Vesicular and vesicopustular eruptions of the hands and feet," Michigan M. Soc. 41:111 1942.

Prognosis is good for cure but prophylaxis is important to prevent recurrences.

Treatment. In the vesicular types 1 to 8,000 potassiumpermanganate packs are useful. In the subacute type Asterol Ointment or the propionic or undecylenic acid fungicides are often effective, while in the chronic type Whitfield's ointment plain or fortified with 0.1 per cent anthralin is generally useful.

DERMATOPHYTIDS

One of the most common complications of an infected or over treated dermatophytosis of the feet, these lesions involve the palms and the fingers. This allergic reaction appears suddenly and usually is preceded by intense itching for 3 or 4 days.

The eruption is usually symmetrical and consists of superficial and deep-seated vesicles or bullae varying in size from 1 mm. to 1 cm. Secondary infection is common (pustules) resulting in pain discomfort and partial disability



FIG. 211 Dermatitis of the hand. Cultures were positive for *Epidermophyton inguinale*.

Localization. The eruption first starts on the side of the fingers then appears on the palms.

Diagnosis. An active irritated focus may be found on the feet or in the groin (*tinea cruris*). Cultures from the vesicles on the hands are sterile. The sequence of events helps to establish the diagnosis.*

Prognosis. From 2 to 3 weeks are required for involution of the lesions, provided that treatment does not cause local sensitization. Recurrences are the rule after each attack of poorly managed fungus infection of the feet.

Differential diagnosis. Pompholyx (neurogenic) may simulate the "id reaction, but there is no active focus of fungus infection present.

Treatment. The active focus on the feet must be treated with hot boric packs to aid drainage and to permit exfoliation of the epidermis. Painting the area with a 2 per cent aqueous solution of gentian violet is safe and useful.

The management of the bullae consists of (1) opening the large ones with a sterile needle or knife blade (2) applying astringent packs (0.5 per cent aluminum acetate) or hot boric acid solution (for vesicopustular eruptions) and (3) as soon as the acute element has disappeared having the patient apply a 5 per cent solution of salicylic acid in alcohol until the lesions desquamate. Antihistamines may be useful to control the itching.

PUSTULAR BACTERIDS

The palms and the soles may rarely present an eruption of vesicopustules resulting from an allergic reaction in a sensitized dermis to an internal focus of infection. The eruption is usually symmetrical and seems to come in crops running through a pustulovesicular stage and later becoming indolent, scaly brownish flat vesicles. During periods of quiescence the lesions may disappear temporarily.

Localization. The thenar and the hypothenar eminences are usually involved. Frequently the eruption also involves the soles to a greater or a lesser extent.

*The subject is discussed further in Lewis, G. M. and Hopper, M. E. An Introduction to Medical Microbiology ed. 3 Chicago, S. Bk. Pub. 1943

Etiology The cause is complex and more factors than merely a focus of infection are necessary for the production of these lesions.

Prognosis. These eruptions are difficult to cure but some observers report cure after removal of infected tonsils.

Diagnosis. The indolent nature of the eruption the history of flare ups following activity of suspected foci and the resistance of the lesion to antibiotic therapy suggests the diagnosis.

Treatment. The suspected focus should be treated or eliminated if the patient can be persuaded * Desensitization with autogenous or stock vaccines and a trial of the antibiotics may be helpful Chloroquine is worth a trial on an experimental basis although its mode of action is not known Local therapy is usually unavailing

PSORIASIS

Poeciatric lesions may be limited to the hands or may exist as part of a generalized eruption. When localized to the hands the lesions are usually atypical, and diagnosis is difficult.

Clinical Appearance. The eruption usually consists of several papulosquamous or hyperkeratotic ovoid or irregular-shaped lesions, covered with a dry hard scale, and is often bilateral. Palmar lesions may have polycyclic borders and are sharply defined. Fissures are common, especially in winter

Diagnosis. This may be difficult in the absence of poeciatric lesions elsewhere. The typical dry silvery scaling may be noted after scraping the lesions. Biopsy if permitted, is characteristic †

Localization. The lesions are bilateral and are found especially on the external surfaces of the joints of the fingers, the finger tips and the middle of the palms at points of pressure.

Etiology Most of the cases occur in men in the 30-year-old to 50-year-old age group. The cause is unknown but the disease may follow trauma, emotional upsets and various physical stimuli

Prognosis. Nonpustular poeciasis is extremely resistant to treatment.

Andrews, G. C. and Machachek, G. F. Pustular bacteridia of the hands and feet, *Arch. Dermat. & Syph.* 52:437 1935

†For details of diagnosis, see Caro, M. R. and Senechal F. E. Psoriasis of the hands, *Arch. Dermat. & Syph.* 56:629 1947

Treatment. Friction, e.g., golf-playing, should be eliminated as far as possible. A tar-anthralin ointment is often effective, but in resistant cases x ray therapy by a qualified expert is indicated.

PUSTULAR PSORIASIS

True psoriasis rarely becomes pustular but this term has been designated for a psoriasiform eruption which presents recurrent pustular lesions. The name is not in good repute but, until replaced by a more definite term, it designates a rare type of eruption.

These lesions are at first ill-defined erythematous macules covered with a dry scale which soon becomes minute superficial sterile pustules. The lesions coalesce to form indolent patches.

Localization. The lesions usually occur on the midpalms and the sides. The soles may be involved at the same time.

Diagnosis is made by a process of exclusion.

Prognosis. The eruption is resistant to therapy.

Etiology is unknown, but focal infection and various metabolic factors may be predisposing.

Treatment is unsatisfactory for cure, but vaccines, antibiotics, sulfa drugs and cortisone may reduce the severity of attacks at one time or another. Local treatment consists of the cautious use of a 3 per cent chrysarobin ointment, 7 per cent tr. iodine or Castellani solution.

FOOD SENSITIZATION ERUPTIONS

There is no agreement concerning the role of sensitization to specific foods in the etiology of dermatoses of the hands. Only when the eruption cannot be classified or when response to all therapy has met with failure, is food sensitivity considered. The type of eruption as well as the localization is variable. Other sites may be involved. The lesions are usually of the patchy vesicular or eczematoid type and are characterized by exacerbations and remissions.

Etiology. The endogenous allergy to specific foods may be primary or a contributory factor. Most of the cases occur in women and especially in those with a history of atopy.

Diagnosis. When all other causes have been eliminated, a his-

tory of unexplained relapses is present, and the examiner has a high index of suspicion regarding food allergy dietary investigation may solve an occasional case if the cooperation of the patient can be obtained. Basic or trial diets are employed. The addition of an allergenic food should cause a flare-up of the pruritus or the eczema within from 15 minutes to several hours. Winston and Sutton* found 8 per cent of their hand eczemas due to food allergy. The typical sites involved were the dorsum of the fingers or the hands or the ulnar surface of the wrists.

Prognosis. Full co-operation must be obtained and only the sustained interest of the physician will be met with success.

Treatment. Local therapy is based on general principles. Starting with a basic nonallergenic diet, a new food is added at 5-day intervals and the effect on the lesions is noted. With intelligent patients a dietary diary may provide a clue. However if the food factor is seriously considered, it should be undertaken only by hospitalizing the patient to avoid error.

*Winston, B. H., and Sutton, R. L., Jr. Dermatitis of the hands due to ingested allergens, *Arch. Dermat. & Syph.* 58:335-343, 1948

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Excerpta Medica 280 Madison Ave. New York 16 N Y

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A.M.A. Archives of Dermatology 535 N Dearborn Street Chicago 10 Illinois.

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